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ORAL SURGERY
ORAL MEDICINE
and ORAL PATHOLOGY

## EDITORIAL

### MAJOR GENERAL JOSEPH L. BERNIER
### UNIVERSITY OF ILLINOIS' OUTSTANDING ALUMNUS

THE Editor is delighted to announce with pride and pleasure that one of our associate editors and editorial consultants has received the Distinguished Alumnus Award from the Dental Alumni Association of the University of Illinois.

Major General Joseph L. Bernier received the award on Nov. 9, 1960, at the University's Professional Colleges Campus of Chicago for outstanding achievements in the field of dentistry. The General is a graduate of the University of Illinois, where he received the Doctor of Dental Surgery degree in 1932 and the Master of Science degree in 1934. In 1959 he was named one of the "Twelve Outstanding Dentists in the History of American Dentistry" by the United States Information Agency and the National Library of Medicine.

General Bernier is the seventh Dental Corps officer to hold the rank of major general, to which he was promoted when he was appointed Assistant Surgeon General of the United States Army on Aug. 1, 1960. He is the first Army dentist to be promoted to major general from the rank of colonel. He also is the first certified oral pathologist in the Army Dental Corps.

General Bernier has made an outstanding contribution to the practice of dentistry by the development of the Oral Pathology Branch of the Armed Forces Institute of Pathology in Washington, D. C., since 1938. This department of the Institute, of which he was chief, has become the largest center in the United States devoted to the diagnosis and study of oral disease. General Bernier also was a leader of a small group of oral pathologists who organized the American Academy of Oral Pathology in 1946, and ten years ago he founded the American Board of Oral Pathology. These developments have been an important factor in placing dentistry on a basic medical foundation, in promoting scientific investigations on a level with other medical specialties, and in elevating the professional standards of dentistry in general.

General Bernier has received many well-earned honors for his widely recognized accomplishments. He shares with two other members of the editorial staff, Lester Cahn and your editor-in-chief, the honor of holding a Fellowship in Dental Surgery in the Royal College of Surgeons, England.

General Bernier has received the Certificate of Achievement in Oral Pathology and Peridontology from the Department of the Army (1946), the Thomas Hinman Award (1955), the John R. Callahan Medal of the Ohio State Dental Association (1955), the Alfred C. Fones Medal of the Connecticut State

*U. S. Army Photograph.*

GENERAL BERNIER.

Dental Society (1957), the Award of Merit from Georgetown University (1957), and the Certificate Award of the Alabama Dental Association (1957). He has received the following decorations: Commandership in the Order of the Crown of Italy, Meritorious Service Medal, Army Occupation Medal (Germany), American Defense Medal, American Campaign Medal, World War II Victory Medal, Korean Victory Medal, European African Middle East Service Medal (two battle stars), and the National Defense Medal. He also has been named an honorary member of many professional associations and societies at home and abroad. Besides, he also is a member of many important committees in Washington. He is a diplomate of the Board of Oral Pathology, the Board

of Oral Periodontology, and the Board of Oral Medicine, and he is Professor of Oral Pathology at Georgetown University School of Dentistry.

General Bernier's most outstanding contributions to the literature include *Differential Diagnosis of Oral Lesions* (Mosby, 1942), *Atlas of Dental and Oral Pathology* (American Registry of Pathology), *The Management of Oral Disease* (Mosby, 1955, 1960), and quite recently *Tumors of the Odontogenic Apparatus and Jaws* (Armed Forces Instute of Pathology). His scientific articles have appeared in our journal as well as in many other dental and medical publications. These have been of great value and interest to the pathologist as well as the practicing dentist and specialist.

We consider Joe to be one of our best friends and supporters. We spent some wonderful days with him and his lovely wife, Bernice, in Washington and abroad. I remember well a dinner at the London Zoo followed by a special tour to see the animals. When a white cockatoo landed on his shoulder—he was a lieutenant colonel then—we expressed the hope that soon it would be an eagle. Now it is two stars. Good luck, Joe, and our heartiest congratulations!

*K. H. T.*

## OPERATIVE ORAL SURGERY

# A RENAL-CELL METASTATIC CARCINOMA INVOLVING THE MANDIBLE AND SUBMAXILLARY GLAND

*Stephen P. Mallett, D.M.D., Boston, Mass.*

A WHITE woman, 72 years of age, presented a swelling of the left submaxillary region of two weeks' duration. The patient stated that she had observed a painful lump which gradually became larger and finally prevented her from opening her mouth wide.

Her past history revealed that she had undergone a cholecystectomy more than thirty years earlier. She had had arthritis in the left knee for many years. There had been no other significant illness, and the family history was irrelevant. The patient had four children who were living and well.

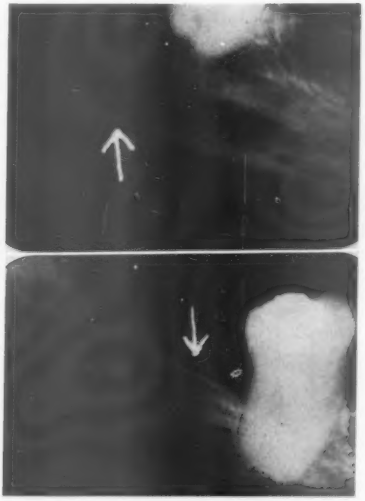

Fig. 1.—Intraoral dental x-ray films showing superior mandibular alveolar border just distal to remaining molar. Arrows point to fracture.

She had a blood pressure of 100/100, temperature of 98° F., pulse rate of 80, and respiration rate of 20. Physical examination yielded negative findings except for the jaw

pain and swelling, and the jaw roentgenograms (Figs. 1 and 2) showed a large, poorly demarcated cystic area of the left mandible, just distal to the second molar, with a pathologic fracture.

Preoperative differential diagnosis was fibrosarcoma, giant-cell tumor, or carcinoma. An exploratory surgical procedure was planned.

Fig. 2.—Lateral-jaw film showing tumor mass. Arrows point to fracture.

The patient was given the usual preoperative medications, including penicillin therapy. Following induction of general anesthesia, a skin incision was made about 1 cm. below the lower border of the left mandible; this was 4.5 cm. long near the angle. Dissection of tissues and muscles was carried out to the external maxillary artery and anterior facial vein, which were identified, ligated, and cut. When exposed, the tumor mass showed complete loss of bone on the buccal aspect and no cystic lining; it appeared pearly gray in color and was fragile, for the most part, without epithelium or periosteum. There appeared to be no clinical separation of tumor tissue from normal tissue, and the tumor mass could not be excised in toto. There was extension into the submaxillary space, and hemorrhage

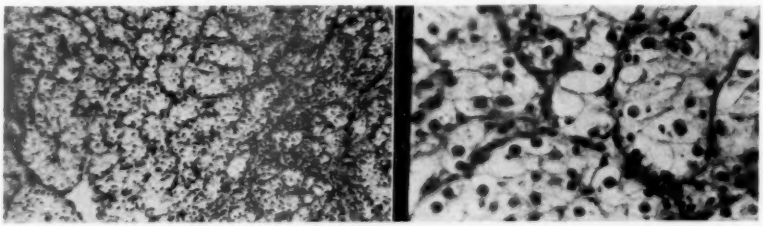

Fig. 3.—Microscopic sections showing clusters of well-differentiated epithelial cells with clear cytoplasm.

resulting from the operation was severe. The lingual plate of bone was somewhat destroyed by invasion of the lesion, and at the pathologic fracture site there was considerable bone loss along the fracture. The internal pterygoid muscle was elevated, the lingual artery was identified and isolated, and small and numerous segments of bone, necrotic-like in appearance, were removed. All visible glandular tissue was excised. Because of the bone loss and the diagnosis of probable malignancy, no attempt was made to reduce the fracture or to place metal bone plates, although the latter were in readiness. The left mandibular second molar

was removed through the extraoral wound; the wound was thoroughly washed with warm saline solution and closed in layers with a rubber band drain.

The pathologist's report was as follows:

*Microscopic Description:* The sections show masses of tumor tissues composed of rough alveolar or tubule-like formations made up of fairly well-differentiated large cells with clear cytoplasm.

*Diagnosis:* Metastatic renal-cell carcinoma (Fig. 3).

Following surgery, an x-ray examination was reported as follows:

Anteroposterior films of the chest and abdomen show considerable osteoporosis, a severe "S" scoliosis, but no destructive metastases. The right costophrenic angle is obliterated by thickened pleura. The heart is slightly dilated (13 to 24.5 cm.). The aorta is small. The lungs are clear.

The lower pole of the right kidney shadow is large. The tips of the catheters are in the renal pelvis. After dye injection on the right, the pelvis is square in shape and slightly enlarged. The lowermost calices are intact but appear to be elevated. No extravasation is noted. The left side is not remarkable.

*Impression:* The findings suggest a tumor of the lower right kidney.

A cystoscopic examination followed. Under 4 per cent Metycaine hydrochloride anesthesia, a No. 24 cystoscope was passed to the bladder, which was entirely normal. Both ureters were catheterized with No. 6 catheters, without obstruction. There was a good but slightly blood-tinged flow from the right and a poor flow from the left. A bilateral pyelogram was suggestive of a possible tumor on the right, but this was not definite. A chest film was taken following this procedure.

The blood urine studies were reported as follows:

*Urine Examinations*

*November 3:* Color—yellow, clear. Sugar—0. Reaction—acid. Specific gravity—1.016. Albumin. Sediment—few epithelial cells, slight hypochromia.

*November 8, Bladder urine:* Color—yellow, clear. Reaction—acid. Sediment—few squamous epithelial cells, occasional white blood cells per high-power field, few uric acid crystals.

*November 8, Right kidney urine:* Color—yellow, clear. Reaction—acid. Sediment—few squamous epithelial cells, few round epithelial cells, 40 to 50 red blood cells per high-power field, bacteria, occasional white blood cells.

*November 8, Left kidney urine:* Color—yellow, clear. Reaction—acid. Sediment—few squamous epithelial cells, 1 to 5 red blood cells per high-power field, occasional uric acid crystals.

*Blood Examination*

Red blood cells—4,000,000. Hemoglobin—10.7 Gm. White blood cells—8,750. Polys. seg.—62. Lym.—31. Monocytes—4. Eosinophils—3. Smear—red blood cells, slight hypochromia. Platelets—normal.

*Serology*

*November 4:* Regular Hinton test—negative.

A urologist made a careful study of this patient's record and roentgenograms and decided against surgery. The patient was discharged to her home on the tenth postoperative day. The extraoral and intraoral sutures and drain were removed on the fifth and ninth postoperative days. The wound and the tooth socket healed uneventfully.

This patient was seen postoperatively at intervals of three, six, and ten weeks, and a satisfactory functional result was evident. Mastication was no problem, although there was a slight left-side pull on opening the mouth. Six months after the operation the patient showed spinal metastasis; she died eight and one-half months postoperatively.

SUMMARY

A case of renal-cell carcinoma metastatic to the mandible and submaxillary region, complicated by a pathologic fracture, has been reported.

47 Bay State Rd.

# PARATHYROID CYST: A PROBLEM IN DIFFERENTIAL DIAGNOSIS

*Morton Malkin, D.D.S.,* Brooklyn, N. Y., and Irving Chapman, M.D.,** Elmhurst, N. Y.*

THE preoperative diagnosis of a mass in the neck is often fraught with difficulty. The following general categories[1] must be considered: acute and chronic regional and systemic infections from a variety of organisms; allergic reactions; primary neoplasms; metastatic malignant disease; malignant lymphadenopathies; sarcoidosis; thyroid, parathyroid, and salivary gland disturbances; vascular aneurysms; and various cervical cysts.

In the classification of cervical cysts, there are six categories[2]: (1) epidermal or sebaceous cysts, (2) branchial cleft cysts, (3) lymphatic cysts or cystic hygromas, (4) thyroglossal duct cysts, (5) papillary cystadenoma lymphomatosum, and (6) cysts with malignant changes. The present article concerns the rare but generally overlooked parathyroid cyst. This is a benign growth, but it can mimic conditions that are much more grave.

At autopsy, microscopic and small macroscopic cysts are occasionally found in the parathyroid glands. However, parathyroid cysts large enough to be seen clinically are rare.[3, 4] A review of the literature since 1951 reveals six papers reporting several proved cases of palpable parathyroid cysts.[3-9] Three other probable cases in which microscopic proof was lacking were reported from the Mayo Clinic.[6] Certain characteristics can be garnered from these cases.[3-9]

Typically, there is a solitary mass in the neck, with no lymphadenopathy. Gradual growth over a period of several months to a few years is usual. If the cyst is large enough, the trachea may be deviated to the opposite side, and enough pressure purportedly may be placed on the recurrent laryngeal nerve to cause hoarseness. On surgical exposure, the cyst characteristically is filled with clear fluid and has a thin, translucent, fibrous wall. On microscopic examination, a fibrous connective-tissue wall is seen, occasionally lined by cuboidal epithelium. Within the wall are groups of two kinds of cells—the chief cells and the oxyphil cells (Figs. 1 to 4). Almost always the cyst has not demonstrated endocrine function.

The proper treatment is surgical excision.[5] These cysts are easily dissected free of surrounding tissues, including the thyroid gland and recurrent laryngeal nerve. Any preoperative symptoms caused by the cyst, such as unilateral vocal cord paralysis,[5, 6] may return to normal in a few weeks.

The following case was observed at our hospital.

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From the Department of Pathology, City Hospital at Elmhurst.
*Assistant Clinical Attending Oral Surgeon.
**Director of Laboratories.

CASE REPORT

*Pertinent History and Physical Examination.*—A 77-year-old woman was admitted to the hospital because of anorexia and general weakness. There was a discrete ovoid mass in the left upper lateral neck below the angle of the mandible and deep to the sternomastoid

Fig. 1.—Cyst wall showing degenerating epithelium at the surface, collagen connective tissue below it, and parathyroid cell clusters within the wall.

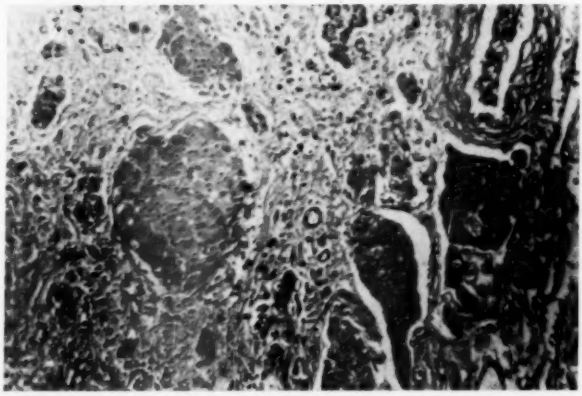

Fig. 2.—Parathyroid cyst with clusters of oxyphil cells at left and chief cells at right.

muscle (Fig. 5). This was firm, nontender, and not freely movable but not fixed to skin. The mass had first been noted by the patient one year before, and it had slowly enlarged to its present size. No lymph nodes were palpable. Aspiration had been attempted two months previously.

*X-ray and Laboratory Findings.*—Alkaline phosphatase was 2.3 Bodansky units. A complete blood count was within normal limits. A chest x-ray showed the trachea deviated to the right; no tumor or lymphomatous infiltration was revealed. The long bones and pelvis gave no evidence of abnormality. The bone marrow was within normal limits.

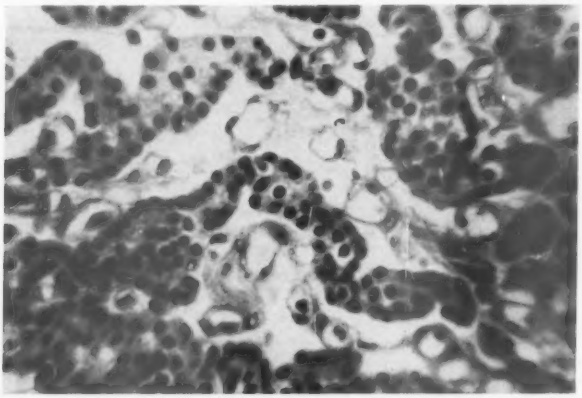

Fig. 3.—High-power photomicrograph of chief cells.

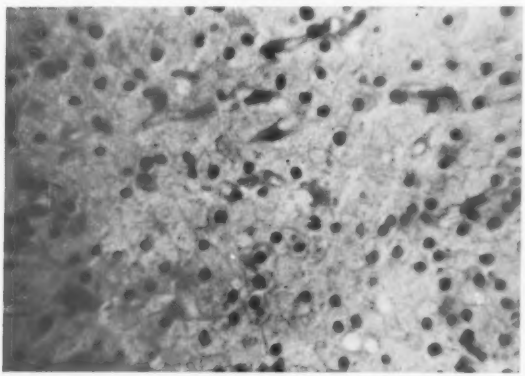

Fig. 4.—High-power photomicrograph of oxyphil cells.

*Hospital Course.*—A clinical diagnosis of branchial cyst was considered most likely. Two weeks after admission, aspiration of the mass yielded a clear, brownish fluid. Because of the patient's poor condition, no surgery was contemplated at that time. The downhill course continued, and the patient died seven weeks after admission. An autopsy was performed and the anatomic diagnoses, other than the cyst in question, were pyelonephritis, pulmonary edema, and recent thrombi in both lower pulmonary arteries.

*Pathologic Description of Cervical Mass.*—

*Gross (Fig. 6):* The mass was ovoid, 7 cm. by 5 cm., completely encapsulated, surrounded by connective tissue, and not adherent to skin. On the upper pole of the mass there was a pedicle, 1 cm. long, which was attached to the posterior and inferior parts of the angle of the mandible. On section, there was a cystic cavity filled with blood and blood clots. The wall was 3 mm. to 5 mm. thick.

*Microscopic (Figs. 1 to 4):* The cyst wall was made up of collagenous, fibrous connective tissue lined in a few areas by a degenerating, flattened epithelium. Within the cyst wall were two types of epithelial cell clusters. One was a light basophilic cell with a distinct nucleus and small amounts of cytoplasm—the parathyroid chief cell. The other was a larger, pale eosinophilic cell with a small, dark-staining nucleus and a greater amount of cytoplasm—the oxyphil cell.

Fig. 5.—Clinical photograph of parathyroid cyst.

Fig. 6.—Gross specimen.

DISCUSSION

Although the parathyroid cyst is rare (some twenty-odd previously reported cases), it should be considered in the differential diagnosis of cervical swellings. Because this cyst is usually situated in the position of one of the lower parathyroid glands, it is often mistaken for a thyroid adenoma.[3] If there are symptoms of a unilateral vocal cord paralysis, it may be mistaken for a thyroid carcinoma.[5, 6] On surgical intervention, however, a translucent, thin-walled cyst

filled with clear fluid and easily separated from surrounding structures will be found. The surgeon should realize that he is probably dealing with a parathyroid cyst and that the proper procedure is local excision.

The etiology is unclear, but there are two theories.[3, 7] One is that these cysts are formed by retention of secretions in the colloid vesicles in the parathyroid gland tissue. The other is that these cysts are derived from glandular epithelial remnants of the third or fourth pharyngeal pouches. The latter theory is held more strongly.

In the cyst reported here, the signs and symptoms were fairly characteristic. The previous aspirations of the mass account for the nontranslucent wall and the bloodlike fluid within.

SUMMARY

The parathyroid cyst is another possibility to be considered in the differential diagnosis of neck masses. The characteristic history, signs, symptoms, and macroscopic and microscopic appearances have been described. A case of an unusually large cyst, proved histologically, has been presented.

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# MAXILLARY AMELOBLASTOMA

*Bernard M. Cohen, D.D.S.,* Far Rockaway, N. Y.*

THE reader is referred to standard texts covering ameloblastoma for details of the various tumor types and their pathologic relationships. For the purposes of this report, it is enough to point out that these lesions do not usually invade soft tissue. Because they invade adjacent bone, it is necessary to remove sufficient healthy bone around the tumor to make certain that all infiltrating islands of cells are excised. Some surgeons feel that cauterization, either chemical or electrical, is equally effective. There is also reason to believe that incomplete removal of tumors at a first operation will result in a more infiltrating recurrent lesion. Some case reports show that carcinomatous recurrences develop after incomplete excision.

In the case to be reported, wide excision into healthy bone was the procedure of choice.

## CASE REPORT

A 52-year-old Negro man was seen on Jan. 30, 1959, with a large swelling in the front of the upper jaw (Fig. 1).

The right maxillary cuspid, the first and second premolars, and the first molars had been removed over a ten-year period. In the early autum of 1957, the patient first noted

Fig. 1.—Intraoral view of tumor mass as seen on Jan. 30, 1959.

*Visiting Oral Surgeon, Morrisania City Hospital; Director, Department of Dentistry and Attending Oral Surgeon, Beth David Hospital; Associate Attending Dentist, St. Joseph's Hospital; Associate Attending Oral Surgeon, Rockaway Beach Hospital.

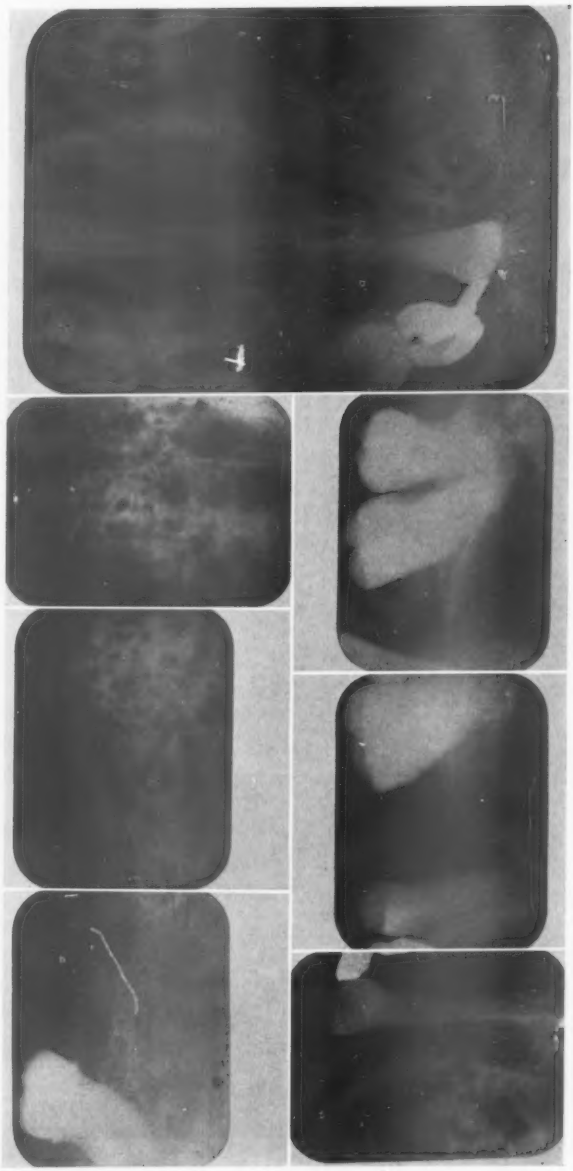

Fig. 2.—Intraoral roentgenograms taken on Jan. 30, 1959, showing multicystic mass.

a swelling in the right maxilla which terminated with the discharge of yellow pus. This repeated itself over the following eighteen months until the winter of 1958, when the swelling enlarged to its present size. In March, 1958, the patient consulted a local dentist who incised and drained the mass. Since March, 1958, the swelling had been painless but had steadily enlarged. There had been a history of right nasal hemorrhage.

*Clinical Examination.*—The patient, a robust man, was in no distress. There was a noticeable enlargement of the right upper lip, produced by a bulging mass in the alveolus (Fig. 1). The intraoral mass was hard and bonelike in consistency, but posteriorly, in the molar area, tenderness and fluctuation were noted. Anteriorly, beneath the right ala of the nose, another tender fluctuant mass was palpated. Speculum examination of the right nostril showed an elevation of the nasal floor. The remainder of the oral mucous membrane was essentially negative.

Fig. 3.—Waters' position roentgenogram showing tumor in antrum.

*X-ray Examination.*—The mandible was normal in all respects. A large reticulated mass was seen in the region of the right maxilla, but there was no evidence of bony invasion or calcification. Intraoral roentgenograms (Fig. 2) of this area showed the mass to be comprised of tiny radiolucent, well-defined areas held together within normal-appearing alveolar bone. The Waters' position film indicated a pronounced clouding of the right antrum (Fig. 3).

*Provisional Diagnosis.*—On the basis of the clinical examination, roentgenograms, and history, a tentative diagnosis of ameloblastoma was made.

*Preoperative Studies.*—The patient was admitted to the hospital on Feb. 5, 1959. The complete blood picture was normal, with the exception of a low white count of 3,600. The blood chemistry, urinalysis, liver profile, and chest x-ray were within normal limits. An electrocardiogram showed a left bundle branch block, with a pulse of about 70 and blood

pressure of 160/100. The remainder of the physical examination was essentially negative, except for a pseudoarthritis which prevented the full extension of the arms. The findings were carefully reviewed by the medical service, and surgery was decided upon.

*Operation.*—On Feb. 10, 1959, the patient was given 3 gr. of Nembutal sodium at bedtime. At 7 o'clock the next morning 25 mg. Demerol hydrochloride and 1/150 gr. atropine were given intramuscularly. The patient also received 600,000 units of penicillin with 0.5 Gm. of streptomycin.

Anesthesia was induced with Pentothal sodium and nitrous oxide—ether—oxygen sequence, and an endotracheal tube was placed. The face was prepared with hexachlorophene, the mouth was cleansed with aqueous Zephiran chloride, and the patient was draped in the manner customary for intraoral operations.

The left maxillary central incisor and cuspid were removed. An incision was then made along the crest of the ridge from the left premolar to the right molar area. A vertical incision was made from the crest of the gingivae into the mucobuccal fold in the left premolar area. The mucosa was reflected anteriorly, until the lesion was encountered. Considerable difficulty was experienced in elevating the mucosa over the anterior bony growth.

Fig. 4.—Antral defect at surgery showing loss of portion of maxilla.

Posteriorly, it was apparent that the mass was cystic. The bone between the two extraction sites was excised to include the lesion and some healthy bone. A chisel was then placed buccally in the region of the left lateral incisor, and the bone was incised. This was repeated in the palatal area and the entire mass was fractured out to the buccal region. Traction on the specimen was continued posteriorly, and the bony mass was seen to be connected to a large cystic area within the right antrum. A quantity of cheesy material was expressed from the torn cystic wall, but it was possible to enucleat the mass en toto. The walls of the antrum were carefully examined for possible encroachment, and a portion of the healthy bone from the buccal and palatal areas was also excised. The antrum was found to be lined with normal healthy mucous membrane, but the antral floor had been deflected by the cystic mass and completely resorbed (Fig. 4).

An opening was made beneath the right inferior nasal concha with a nasal rasp. The anterior extension of the cystic tumor in the floor of the nose had eroded the thin bone beneath the inferior nasal concha, so that only the mucous membrane of the nose remained. The antrum was packed with 1 inch iodoform gauze, the end of which led out through the opening in the nasal wall. The wound was then approximated, but it was necessary to remove some of the soft tissue which had covered the anterior tumor mass. It was felt that this tissue might have been seeded by some of the tiny microcysts, even though it is thought that ameloblastoma does not invade soft tissue in this manner. The mucous membrane was then

closed with 000 silk in such a way that the ridge continuity was preserved. The size of the tumor mass was approximately 6 by 3 by 2 cm.

The patient was returned to his bed and reacted to his immediate surroundings. By noon all vital signs were normal, and the patient had fully reacted but was somewhat drowsy. At this time 50 mg. of Demerol hydrochloride was given to control pain, but no other narcotics were found necessary. Equanil therapy was prescribed. The second and third postoperative days were not remarkable, except for the patient's complaint of headache and sore throat. The headache was a result of the antral packing, while the sore throat was caused by the endotracheal tube.

*Pathologist's Report.*—The specimen consisted of an irregularly shaped mass which was reddish brown in color, partially cystic, and measured 8 by 2.5 by 2 cm. One surface was indurated and presented a fine, granular, brownish yellow surface. The opposite surface was soft, elastic, and membranous. Attached to the mass were several irregularly shaped pieces of yellowish brown tissue measuring approximately 3 by 3 by 0.5 cm.; the tissue was firm and elastic with multiple areas of calcification.

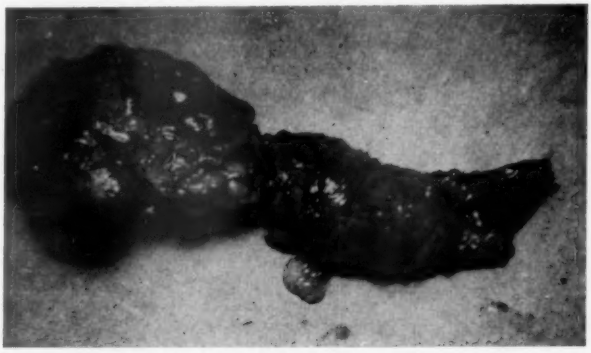

Fig. 5.—Tumor mass with portion of palate and sinus.

*Microscopic:* The section taken from the oral mucosa showed dense fibrous tissue partially covered by squamous epithelium. In areas there was surface erosion with exposure of the underlying fibrous stroma. The subepidermal tissue showed diffused hemorrhage and infiltration by scattered leukocytes and lymphocytes. In close proximity to the surface epithelium, there were cystic spaces in the stroma; these were lined with flattened cuboidal epithelium, to which were attached well-organized intracystic squamous islands. The surface of these islands showed a palisading arrangement of cuboidal cells with perpendicularly arranged ovoid nuclei. These cells merged imperceptibly with underlying squamous cells which displayed intercellular bridges, keratinization of the epithelial cells, and keratin pearl formation. Within these squamous islands there were scattered cystic spaces lined with cuboidal epithelium and filled with eosinophilic-staining fibrillary structures of varying condensation. Elsewhere within the fibrous stroma there were scattered epithelial islands composed of cuboidal epithelium. There were also fragments of degenerated bone trabeculae showing areas of osteoid bone formation. The fragment of this bone tissue was connected with a rather dense epidermoid tumor mass, similar in appearance to the ones described previously. Also included into this section was a hemangiomatous area composed of thin-walled capillary tissue with intense engorgement (Figs. 5 and 6).

*Diagnosis:*

(1) Microcystic ameloblastoma, acanthoma type. (2) Ameloblastoma with capillary hemangioma.

*Postoperative Course.*—The fourth, fifth, and sixth days were uneventful. The patient did not receive any narcotics after the initial dose of Demerol hydrochloride. On the tenth

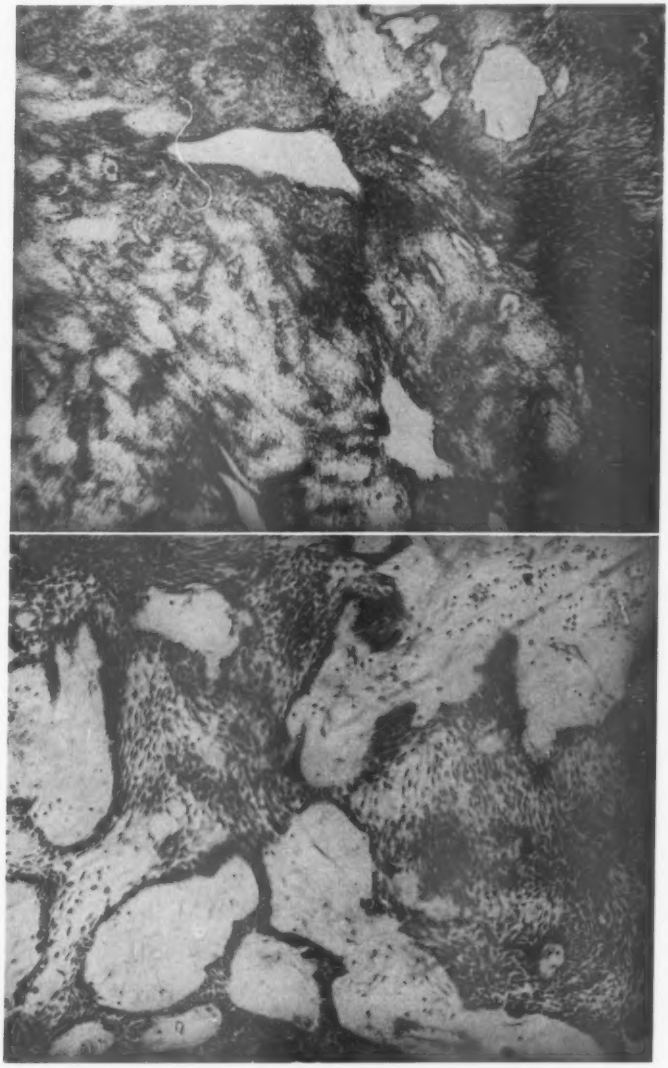

Fig. 6.

day the nasal packing was removed. The patient was discharged on the twelfth postoperative day, to be followed in the outpatient department. At the time of discharge, the oral wound had healed remarkably and the sutures were removed.

## CONCLUSION

A case of maxillary ameloblastoma in which the treatment of choice was wide surgical excision has been presented. The patient was followed for a while

Fig. 7.—Intraoral view of mouth on May 30, 1959, showing healed site of surgery.

Fig. 8.—Waters' position roentgenogram taken on May 30, 1959, showing aeration of the antrum.

after a prosthesis had been constructed to restore normal function and appearance. To date there is no evidence of recurrence, but observation for a number of years is necessary in all such cases.

1927-31 Mott Ave.

## EXODONTIA

# CORRECTION OF CLASS I MALOCCLUSION WITH SEVERELY TRAUMATIZED INCISORS

### Report of a Case

*Louis B. Kelsten, D.D.S., Newark, N. J.*

On Nov. 3, 1956, a 14-year-old boy came to my office for routine orthodontic consultation. Because he had a Class I malocclusion with crowded upper and lower incisors, I recommended that the four first premolars be extracted. Since the boy's parents opposed the extractions, such records as photographs, roentgenograms, and models were not made.

A week later, I received an emergency call from the patient's family asking me to see the boy immediately. While the boy was engaging in a one-sided fight that morning with his older brother, his upper right lateral incisor had been knocked out; the upper right and left central incisors hung in a clot of blood, and the upper left lateral incisor was loose (Fig. 1). Because the boy was severely depressed at the prospect of losing his remaining upper incisors, and because his parents pleaded with me to save those teeth, I promised to see what I could do.

An occlusal film (Fig. 2) revealed that there were no fractured teeth. The parents were advised that if the teeth were splinted and normal healing of the supporting tissues took place, the boy would be referred to an endodontist for root canal therapy in order to salvage as many anterior teeth as possible. Later, orthodontic treatment could be undertaken to correct the occlusion and tooth alignment.

At this visit an upper splint (Fig. 3) was made, care being taken not to dislodge the badly loosened teeth. Two stainless steel bands were made for the upper first premolars with strips of band material welded labially, buccally, lingually, and across incisally to house quick-setting zinc oxide and eugenol cement. Fig. 4 shows the splint cemented into position with quick-setting zinc oxide and eugenol cement.

While the upper splint was being fabricated, an alginate impression of the lower jaw was made and poured with quick-setting stone. After separation of

Fig. 1.—Condition of patient's mouth following fight with his brother. Note injury to supporting tissues and displacement of the remaining upper incisors.

Fig. 2.—Occlusal film taken on Nov. 10, 1956.

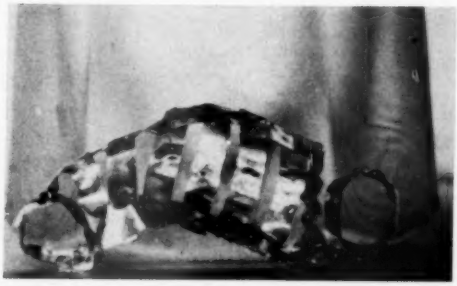

Fig. 3.—Stainless steel appliance used as a splint for the upper incisors.

the model, a bite plate of self-curing acrylic with two molar clasps was made. Fig. 5 shows the lower bite plate which covered the occlusal surfaces of the lower molars and premolars in order to open the boy's bite so that during mastication he would not contact the upper splint. Fig. 6 shows the upper and lower appliances in position. At first the boy was placed on a soft diet, which was changed in four weeks to a semisolid diet.

About three months (Feb. 7, 1957) after the upper splint was removed, the supporting tissues appeared in good health and the teeth were firm and responded to vitality tests (Fig. 7). At this time the parents were told that to eliminate the need for an upper anterior bridge, the upper lateral space should be closed.

Fig. 4.—Upper splint cemented into position with quick-setting zinc oxide and eugenol cement. Note blood oozing from the fractured labial alveolar process.

Fig. 5.—Lower bite plate used to open bite and thereby protect upper splint during mastication.

Also, to balance the tooth material of both jaws so that the patient could have a fairly good occlusion and tooth alignment, the lower right central incisor should be extracted. To this they consented.

On Feb. 28, 1957, two weeks after the lower incisor was extracted, bands were cemented on the upper and lower molars, premolars, cuspids, and incisors. Then 0.016 inch leveling arches were placed. In three weeks these were replaced with 0.018 inch arches. After the teeth were uprighted and rotated into a better occlusion, 0.019 by 0.026 inch upper and lower arches with Bull loops were inserted. These loops were activated every three weeks until the spaces were closed.

Following this, the cuspid and premolar bands on both jaws were removed except for those on the upper right cuspid, and 0.021 by 0.027 inch arches with coils just mesial to the molar tubes were used to eliminate any remaining di-

Fig. 6.—Upper and lower appliances in position.

Fig. 7.—Following removal of the upper splint. Note healed supporting tissues; the teeth appear firm, and they responded positively to vitality tests.

Fig. 8.—Final upper and lower orthodontic appliances in position revealing closure of spaces, alignment of the teeth, and corrected occlusion.

astema between the teeth. Then the 0.021 by 0.027 inch arches were left in position for two months (Fig. 8). On Oct. 2, 1957, all appliances were removed. No retainers were used.

Fig. 9 shows the completed case on Oct. 10, 1958, one year after the appliances were removed.

Before the patient was dismissed, the parents were told that the upper incisors required observation for several years since traumatized teeth can become nonvital in two to five years. (Note: Because the patient enlisted in the Navy, he has not been seen since October, 1959. At that time his incisors were still vital.)

Fig. 9.—The completed case one year after removal of appliances.

In conclusion, since the loss of upper anterior teeth can cause psychologic disturbance as well as badly complicate malocclusions in children, severely traumatized incisors should be salvaged if at all possible.

31 Lincoln Park.

# ORAL MEDICINE

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## INFANTILE CORTICAL HYPEROSTOSIS

### Report of a Case

*Leonard Szerlip, B.A., D.D.S., and Alvin R. Mintz, A.B., M.D.,*
*Morristown, N. J.*

WE FIRST saw the patient, a 3-month-old girl, on Nov. 4, 1958, when a consultation was requested by the pediatrician who was attending her. The hospital chart revealed that she had been admitted on November 1 with the diagnosis of "swollen left mandible."

*History of Present Illness.*—One week prior to admission, the patient suddenly developed a swelling of the lower left eyelid. Swelling of the left jaw followed within one day. The mother described the swelling as "stony-like." The only other symptom was a concurrent loss of appetite. A local physician referred the infant to a pediatrician, who hospitalized her.

*Past History.*—The infant's birth was uneventful, with the elective use of low forceps at term. Except for a minor feeding problem, the patient had been in good health.

*Family History.*—The parents were living and well. There were three siblings—two brothers and a sister—all in good health.

*Roentgenographic Examination.*—The chart contained two roentgenographic reports made at the time of initial examination. On November 1 the roentgenologist reported: "Radiographic examination of lower jaw shows no evidence of an intrinsic osseous disease process. We must emphasize the fact that the patient's age prevented accurate positioning and technique." A report on November 4 said: "Radiographic examination of the long bones failed to reveal any evidence of a primary or secondary intrinsic osseous disease process. No significant congenital anomalies are detected."

On consultation, we asked that roentgenographic examination of the jaw lesion be repeated. The report, dated November 5, follows: "Multiple views of the face disclose the maxillary bone to be symmetrical on either side. There is a thickening of the soft tissues which partially obscure the left maxilla. On the base view, the left mandible in its lateral margin is expanded. There is no sclerosis or periosteal reaction to suggest either an osteoma or an infantile cortical hyperostosis. The diagnostic possibilities include fibrous dysplasia, or possibly a giant-cell tumor of the mandible, a so-called epulis."

*Laboratory Findings.*—Temperature on initial examination was 99° F. The range for the four days, as recorded on the chart, was 98 to 99.4 degrees. Pulse and respiration were normal. Examination of the blood revealed 9.60 Gm. per cent hemoglobin, a hematocrit of 30 per cent, a white blood cell count of 18,500 per cubic millimeter with a differential count of 45 per cent neutrophils, 59 per cent lymphocytes, and 1 per cent monocytes; the

Presented at the March 30, 1959, monthly conference of the New York Institute of Clinical Oral Pathology held at the New York Academy of Medicine.

absolute eosinophil count was 110 per cubic millimeter. Urinalysis was within normal limits. Blood chemistry studies revealed 9.85 mg. per cent calcium, 2.9 mg. per cent phosphorus, and alkaline phosphatase 2.7 Bodansky units.

*Clinical Examination.*—Marked asymmetry of the face was noted, with distortion of the left side (Fig. 1). There was a small, hard (bonylike) swelling in the left infraorbital region. A large, hard (bonylike) swelling of the entire left ascending ramus of the mandible was present. This latter swelling could be palpated intraorally on the lateral surface of the ascending ramus, and it obliterated the buccal sulcus in the retromolar area. The overlying skin was of normal color and texture, as was the overlying mucosa intraorally. The area apparently was not tender, as the baby did not vigorously resist examination.

Fig. 1.—Although there was some movement which caused blurring in this photograph, the enlargement of the left side of the face is quite evident.

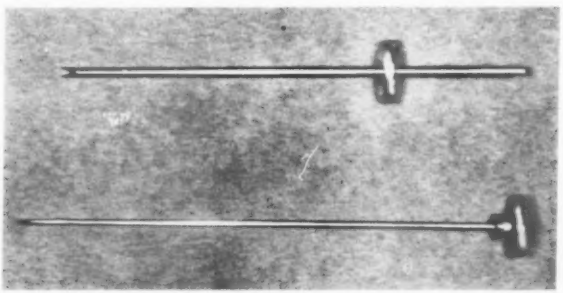

Fig. 2.—Ellis biopsy drill (above) and its stylet (below). This instrument is used in a dental handpiece for biopsy of hard structures.

In the consultation report the clinical findings were noted, and it was concluded that, since there were no signs or symptoms to support the diagnosis of an inflammatory process, we were probably dealing with a rapidly growing neoplasm, probably a malignancy. A biopsy was recommended in order to establish a diagnosis.

*Biopsy Procedure.*—After a general anesthetic had been administered, an incision was made intraorally along the anterior margin of the ascending ramus from approximately the base of the coronoid process, downward and forward along the crest of the alveolar ridge,

to the area of the mental foramen. The mucoperiosteum was stripped laterally and the flap was retracted. This procedure resulted in extrusion of the bud of the deciduous second molar, and this was removed. The bone on the lateral surface of the mandible, though markedly distended, showed no erosion or other abnormalities. An Ellis biopsy drill (Fig. 2) was used to remove two specimens at different levels from the ramus. The drill was passed in an obliquely posterior direction, care being taken to avoid the mandibular vessels. The wound was closed with 0000 black silk interrupted sutures.

On November 10 the pediatrician put the baby on Aristocort, 1 mg. twice a day, on an empirical basis. This medication was continued for the duration of the hospital confinement and for twenty days following discharge.

Following the surgical intervention, the baby seemed to improve rapidly. The swelling had seemed to increase daily, but immediately following the biopsy procedure it appeared to regress fairly markedly. The baby began taking feedings better and generally seemed more content.

The pathology report indicated "fibrous dysplasia with bone resorption." This appeared to be incompatible with the history and clinical findings. Since the patient had improved so markedly, it was suggested that she be discharged and followed in the office. Accordingly, she was discharged from the hospital on Nov. 20, 1958.

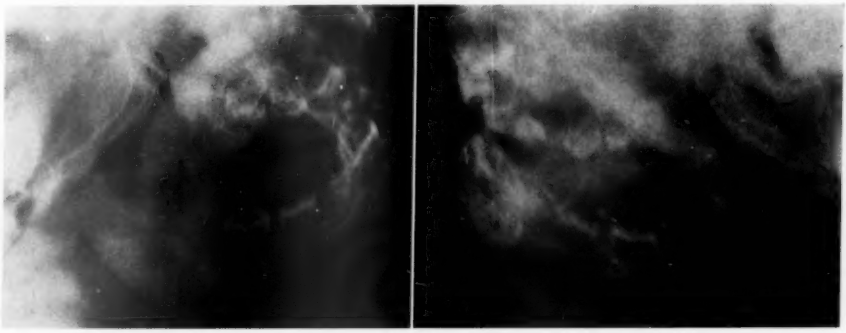

Fig. 3.—Left and right sides of mandible. Comparison of the rami points up the characteristic roentgenographic appearance of infantile cortical hyperostosis on the left side.

On November 25 the infant was seen in the office, where additional and what were thought to be more diagnostic x-ray films were taken (Fig. 3). The roentgenograms and a slide of the biopsy specimen were submitted to Dr. H. L. Jaffe at the Hospital of Joint Diseases in New York City. He suggested the diagnosis of infantile cortical hyperostosis, and this was further confirmed by the New York Institute of Clinical Oral Pathology as well as by the Armed Forces Institute of Pathology.

The microscope description as reported by Dr. M. Freund of the New York Institute of Clinical Oral Pathology, Inc., follows:

> *Microscopic:* The section shows a framework of newly formed bone trabeculae covered by osteoblasts and in many areas still showing osteoblastic cells within the matrix. This is surrounded by loose connective tissue which also fills the intertrabecular spaces. On one aspect, which is its periosteal portion, the connective tissue is dense and shows a good deal of osteoid, and in the depths the trabeculae are sparse and loose connective tissue predominates. Moderate infiltration by plasma cells in this area and, in the denser portion, by polymorphonuclear leukocytes is seen. In occasional areas osteoclasts are present.

When the patient was seen on Feb. 25, 1959, only a very slight palpable swelling of the ascending ramus was still present, and roentgenographic examination disclosed only minimal evidence of the disease (Fig. 4).

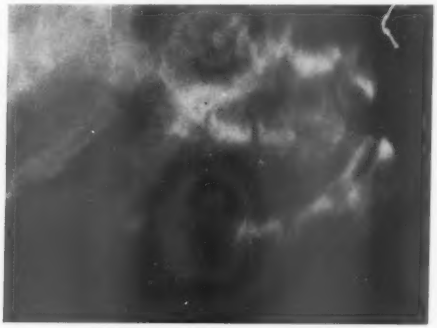

Fig. 4.—Film of left side of mandible taken three months postoperatively. Compare wth Fig. 3.

## DISCUSSION

Infantile cortical hyperostosis (Caffey's syndrome or disease) was first reported in the dental literature in 1958.[1, 2] It should be emphasized that the diagnosis is primarily roentgenographic and not microscopic.

Caffey is of the opinion that there is no valid case of infantile cortical hyperostosis in which the onset occurs later than the fifth month of life. He states that there are but three manifestations which are common to all patients: hyperirritability, swelling of the soft tissues, and cortical thickening of the underlying bones.[3] The most constant laboratory findings are an increased sedimentation rate of erythrocytes and an increased phosphatase activity of the blood serum.[3] Caffey further affirms the fact that fever may be absent when the disease is limited to the mandible, and in the mild facial form the swelling may disappear in a few weeks.[4]

The adrenocorticosteroids are highly effective in the treatment of the disease. "Recurrence is likely when the cortisone is stopped at the end of one or two weeks. Dosage should be reduced gradually to prevent rebound phenomena which have been severe after sudden stoppage of the steroid."[4]

## SUMMARY

We have presented a case of infantile cortical hyperostosis in which the diagnosis was not arrived at immediately because of lack of familiarity with the disease by the clinicians and the pathologist involved. The incompatibility of the original microscopic diagnosis with the clinical picture led to consultation and a correct diagnosis. This case is noteworthy because of the extremely mild form in which the disease occurred, with clinical and roentgenographic evidence showing recovery nearly complete within four months.

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10 PARK PLACE.

# RECURRENT APHTHOUS ULCERATIONS IN A PROFESSIONAL SCHOOL STUDENT POPULATION

## IV. Twelve-Month Study of Natural Disease Patterns

*Irwin I. Ship, D.M.D.,* Alvin L. Morris, D.D.S., Ph.D.,** Roy T. Durocher, D.D.S.,*** and Lester W. Burket, D.D.S., M.D.,*****
*Bethesda, Md., and Philadelphia, Pa.*

### INTRODUCTION

THE need for developing tools for the clinical and epidemiological investigations of oral mucosal diseases is best illustrated by a brief review of the multiple clinical impressions reported in the recent literature regarding these diseases. With regard to recurrent aphthous ulcerations, a disease characterized by recurrent formation of ulcerations in the oral mucosa, there has been a lack of information about the natural patterns of disease in a non-hospital or non-clinic population.

Recurrent aphthous ulcerations have been etiologically associated with hypersensitivity, endocrine disturbances, trauma, avitaminosis, environmental stresses, and emotional problems on the basis of both individual and group reports.[1-6] The clinical association of recurrent "canker sores" with gastrointestinal, dermatologic, ophthalmologic, and other system diseases has been previously suggested.[7-11]

In the first three reports in this series, investigations of this disease in a professional school student population were described. The prevalence, associated medical history factors, and oral examination findings were analyzed to reveal possible clues to etiology.[12-14] However, questionnaire data collected from this population would not answer several important questions regarding this disease. Would a history of recurrent aphthous ulcerations be an accurate indicator of disease severity and status when compared with actual follow-up data? If, as suggested by history, there is a relationship between "canker sores" and the menstrual cycle, particularly in girls and women with severe disease (more

*Clinical Investigations Branch, National Institute of Dental Research, United States Public Health Service, Department of Health, Education and Welfare, Bethesda, Maryland. Present address: Dental Research Laboratory, Philadelphia General Hospital, Philadelphia, Pennsylvania.

**Associate Professor of Oral Medicine, School of Dentistry, University of Pennsylvania, Philadelphia, Pennsylvania.

***Assistant Professor of Oral Medicine, School of Dentistry, University of Pennsylvania,

****Dean, School of Dentistry, University of Pennsylvania.

than one new lesion per month), which phase of the menstrual cycle demonstrates the greatest influence? Do variations in severity of the disease follow natural seasonal patterns? Do examination and vacation periods in the student's life affect this disease? And what are the relationships of severe and mild disease patterns to these environmental stress and unstressed situations? Do male and female patients follow different patterns of disease, as previously suggested, or were those findings a result of natural selection?

It is clear that these questions can be answered accurately only through careful documentation, over an extended period of time, of all individual ulcers in a population with recurrent aphthous disease.

The purpose of this study was to analyze the above parameters of disease in such a population over a twelve-month period.

## MATERIALS AND METHODS

On the basis of questionnaire data submitted by medical history information, 1,788 students attending the University of Pennsylvania Schools of Medicine, Dentistry, Veterinary Medicine, Dental Hygiene, and Nursing and the Schools of Nursing of the Philadelphia General Hospital, the Presbyterian Hospital, and the University of Pennsylvania Hospital were assigned to the following groups: severe (more than one new ulcer per month), mild (less than one new lesion per month), and those with a negative history of recurrent aphthous ulcerations.

MONTHLY DIARY MARCH, 1959

| SIGNS & SYMPTOMS | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TOTAL ULCERS | 0 | 1 | 1 | 2 | 2 | 1 | 1 | 1 | 2 | 2 | 3 | 3 | 3 | 4 | 4 | 3 |
| LIPS | | A | A | A | A | A | A | | | | | | | B | B | B |
| CHEEKS | | | | | | | | A | A | A | AB | AB | AB | B | | |
| TONGUE | | | | A | A | | | | B | B | B | B | B | B | BC | C |
| GUMS | | | | | | | | | | | | | | A | A | A |
| OTHER | | | | | | | | | | | | | | | | |

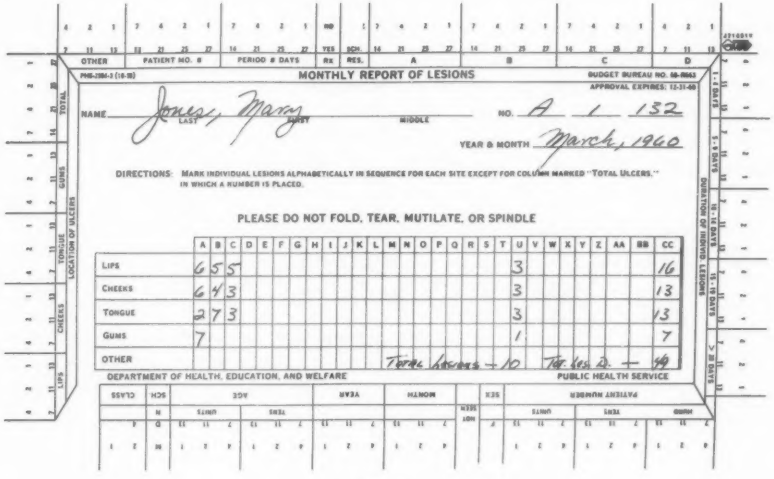
| SIGNS & SYMPTOMS | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| TOTAL ULCERS | 3 | 3 | 2 | 2 | 1 | 1 | 0 | 0 | 0 | 1 | 1 | 1 | 0 | 0 | 0 | |
| LIPS | B | BC | C | C | C | C | | | | | | | | | | |
| CHEEKS | | | | | | | | | | C | C | C | | | | |
| TONGUE | C | | | | | | | | | | | | | | | |
| GUMS | A | A | A | A | | | | | | | | | | | | |
| OTHER period | | X | X | X | X | | | | | | | | | | | |

PHS-2580-2 (REV. 10-58)

Fig. 1.

Three hundred one of the students in "mild" and "severe" groups were given oral examinations.[14] This represented 50 per cent of the total "mild" and "severe" population. Following oral examinations, each student was instructed in the maintenance of diary forms, and all aphthous lesions were documented on Keysort diary cards according to a standardized schema (Figs. 1 and 2). Each lesion was recorded by location in the mouth on each day of its presence

and was assigned an alphabetical code letter to distinguish it from other ulcerations. The letter "A" was assigned to the first lesion for the month, "B" for the second, and so forth through the alphabet for each location; each letter was inserted in the appropriate box for the entire duration of the lesion. The initiation of any lesion was based on the presence of pain or tenderness and the observation of a grayish yellow crateriform interruption in the mucosal tissues. The disappearance of the lesion was determined by absence of pain and the resolution of the grayish yellow crater with the reconstitution of the mucosal integrity. Days of menstruation were indicated by female participants. Periods of examination and vacation for each class were checked with the various schools involved and indicated on a master calendar. Diary cards were collected on a month-to-month basis for a fifteen-month period, starting Jan. 1, 1959. Close working relationships with individual students were facilitated by direct contact.*


MONTHLY REPORT OF LESIONS

BUDGET BUREAU NO. 68-R663

APPROVAL EXPIRES: 12-31-60

NAME Jones, Mary (LAST, FIRST, MIDDLE) NO. A 1 132

YEAR & MONTH March, 1960

DIRECTIONS: MARK INDIVIDUAL LESIONS ALPHABETICALLY IN SEQUENCE FOR EACH SITE EXCEPT FOR COLUMN MARKED "TOTAL ULCERS," IN WHICH A NUMBER IS PLACED.

PLEASE DO NOT FOLD, TEAR, MUTILATE, OR SPINDLE

| | A | B | C | D | E | F | G | H | I | J | K | L | M | N | O | P | Q | R | S | T | U | V | W | X | Y | Z | AA | BB | CC |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| LIPS | 6 | 5 | 5 | | | | | | | | | | | | | | | | | | 3 | | | | | | | | 16 |
| CHEEKS | 6 | 4 | 3 | | | | | | | | | | | | | | | | | | 3 | | | | | | | | 13 |
| TONGUE | 2 | 7 | 3 | | | | | | | | | | | | | | | | | | 3 | | | | | | | | 13 |
| GUMS | 7 | | | | | | | | | | | | | | | | | | | | 1 | | | | | | | | 7 |
| OTHER | | | | | | | | | | | | | | | | | | | | | Total Lesions → 10 | | | | | | | | Tot. Les. 2 → 49 |

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE — PUBLIC HEALTH SERVICE

Fig. 2.

## RESULTS

A total of 230 students (120 males, 110 females) submitted diary forms at monthly intervals of periods of from three to fifteen months. This represented 77.2 per cent of the total avaliable population of 298 students with mild and severe disease (three students had dropped out of school in the intervening period). The mean age of male students was 23.4 years as compared with 18.8 years for the female students. In all aspects, this population demonstrated no significant differences from the original population. The ratio of male to females students and "severe" to "mild" subjects remained unchanged throughout the period of study.

*The authors wish to express their gratitude to Mrs. Olivia Richardson for performing this service.

*Comparison of Actual and Expected Severity of Disease.*—A close correlation between the students' initial report of the frequency of ulcerations and the actual performance over the twelve-month period was noted (Fig. 3). Differences between male and female subjects in this correlation were not seen, and these relationships were observed in all analyses.

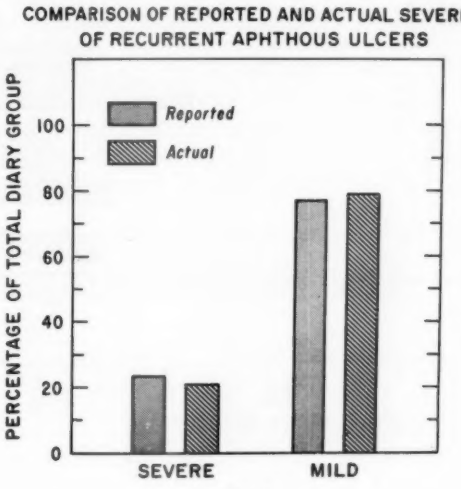



Fig. 3.

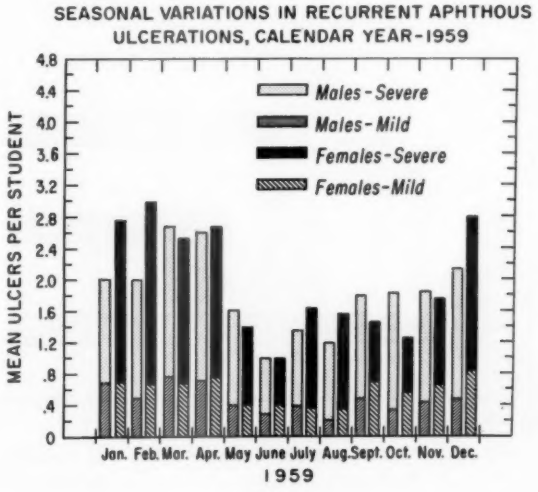



Fig. 4.

*Seasonal Variations.*—For both the "severe" and "mild" groups, large variations in disease severity status were observed over the twelve-month period

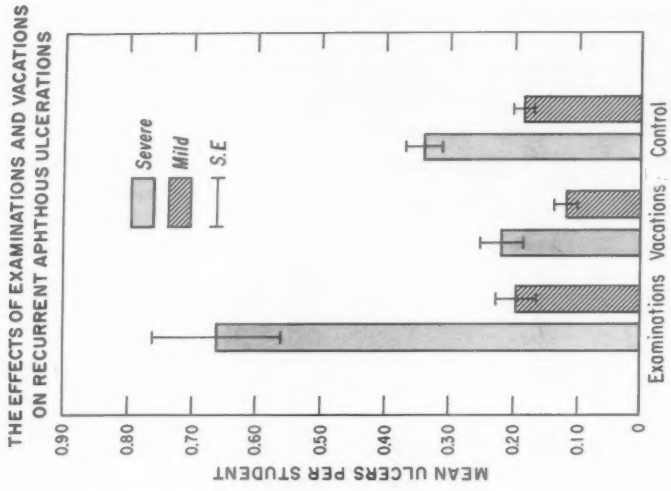

THE EFFECTS OF EXAMINATIONS AND VACATIONS ON RECURRENT APHTHOUS ULCERATIONS
MEAN ULCERS PER STUDENT
0.90
0.80
0.70
0.60
0.50
0.40
0.30
0.20
0.10
0
Severe
Mild
S.E
Examinations
Vacations
Control

Fig. 6.

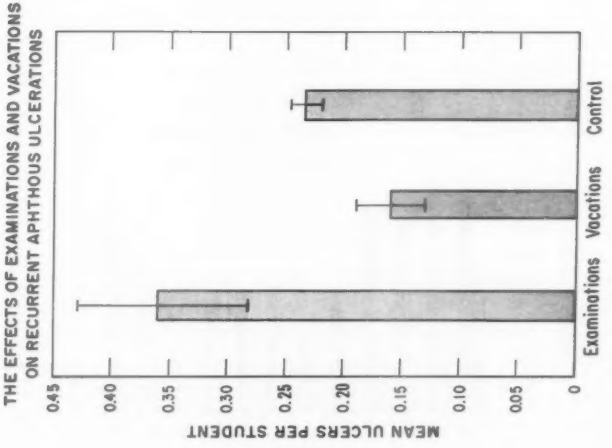

THE EFFECTS OF EXAMINATIONS AND VACATIONS ON RECURRENT APHTHOUS ULCERATIONS
MEAN ULCERS PER STUDENT
0.45
0.40
0.35
0.30
0.25
0.20
0.15
0.10
0.05
0
Examinations
Vacations
Control
S.E. = |—|

Fig. 5.

(Fig. 4). Increased frequency of lesions in the winter months and relative remission during the summer months were observed in all groups, although the "severe" group demonstrated greater proportional changes. Differences between sexes were not significant because of large variations in each group for each month.

*The Effect of Examinations and Vacations.*—The responses of all students to examination and vacation periods as compared to normal school periods are seen in Fig. 5. This suggests increased disease activity during periods of examinations and decreased disease activity during periods of vacation as compared with weeks of normal school activities. Fig. 6 demonstrates that these responses are significant in the groups with "severe" disease and of questionable significance in the group with "mild" disease. Fig. 7 indicates that male students respond in a more dramatic fashion than female students.

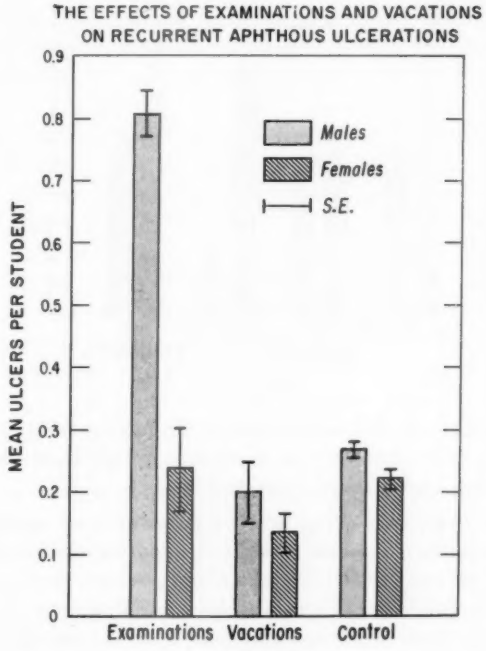



Fig. 7.

*Effects of the Menstrual Cycle.*—The mean ulcers per female student for each of four seven-day periods within the menstrual month are seen in Fig. 8. Week 1 represents the seven days starting on the first day of the menstrual period, and Week 2 represents the following seven days. Week 3 consists of the seven days following the fourteenth day preceding the first day of the succeeding period, and Week 4 starts on the seventh day preceding the first day of the succeeding menstrual period. Although not strictly accurate, these

periods were designated "menstruation," "postmenstruation," "ovulation," and "premenstruation." As indicated, students with more than one lesion each month demonstrated a distinct pattern characterized by exacerbations during the premenstrual week and relative remission during the postmenstrual weeks, with the weeks of menstruation and ovulation intermediate and essentially the same.

In the milder group, in which less than one new lesion appeared monthly, the menstrual and premenstrual weeks were similar and relatively high, while postmenstrual and ovulatory weeks represented considerably less activity.

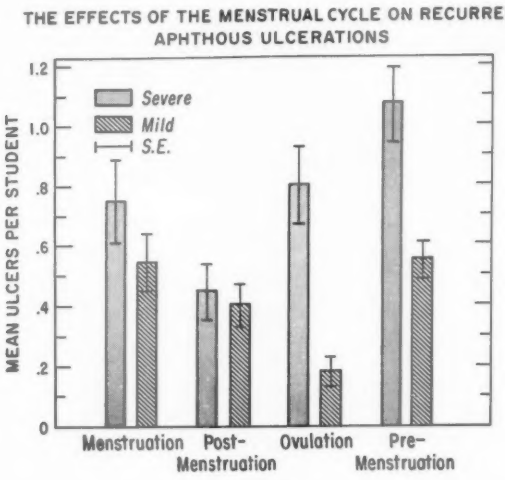



Fig. 8.

*Location of Lesions.*—The distribution of lesions by location in the mouth was essentially as previously reported from oral examination data.[14] Sex and disease severity did not affect these relationships.

*Other Indices of Disease.*—The mean number of new lesions per student appeared to be the index of disease severity for the various analyses performed during the course of this study. However, analyses of mean lesion days per student (the number of lesions times the days per lesion) and mean days per lesion were also computed. The mean ulcer days per student did not differ from the mean lesions per student in any analyses. This was due to the fact that mean days per lesion remained constant throughout.

## DISCUSSION

As previously reported, comparison of the severity of disease by history was closely correlated with the activity seen during oral examinations.[12, 14] Likewise, the history of severity and the findings on oral examinations, as interpreted by the incidence of new lesions developed, correlated closely with

the actual severity as seen through analyses of records extending over twelve months of continued observation. Thus, it would appear that information concerning chronic, recurring oral lesions solicited from populations favorably oriented toward the healing arts has considerable value in the investigation of oral disease.

It is interesting to note the dramatic seasonal variations in disease status that were observed. That these variations were independent of other variables in the study, such as menstruation, severity of disease, and sex, is clear, as the latter remained the same throughout the year. In addition, the academic schedule was such that distributions of examinations, vacations, and normal school periods were unaffected by seasons among the nursing school students. The male subjects (medical, dental, and veterinary school students) had major examinations in December and April, during which several major examinations were concentrated within one-week periods. Thus, environmental influences that were specifically analyzed in this study also failed to demonstrate any influence on seasonal variations. Examination of the relationship of school calendar to the menstrual cycle in female students failed to yield any additional information.

It is logical to expect that drug investigations initiated during one season and completed during another might indicate false results, either masking a true effect if started during the summer and completed during the fall and winter, or showing a false effect if started during the winter and spring and finished during the summer when natural remission appears to occur. Thus, investigations of the effects of therapeutic agents on recurrent apthous ulcerations should be carried to completion within the same season if possible.

As previously reported, strong correlations were noted between a history of emotional factors and a history of recurrent apthous ulcerations.[13] The severity of disease was reported to be directly related to the magnitude of these factors in all analyses as reported by medical history. That environmental changes, such as examinations and vacations, in the school calendar have an effect on disease activity has also been reported previously; and this is again seen in this study. It is interesting to note that male students responded to a greater degree to examinations than did the female students. This could possibly be due to the concentrated nature of the examination schedules in the medical, dental, and veterinary schools. The students with severe disease responded with greater changes as compared with the group with mild disease. This confirms our previous analyses in which 21.4 per cent of students with severe disease reported an association of lesions with nervousness preceding examinations, while 4.9 per cent of students with mild disease reported this association.

That reduced activity is seen during vacation periods would suggest that anxiety and fear associated with major examinations exert an influence on disease activity, particularly in relation to the severity of illness. General physical fatigue, however, might also play a role in these differences. This

was noted in the analyses of medical histories, in which 22.4 per cent of the population with severe disease associated exacerbations with fatigue while 12 per cent of the group with mild disease reported this association.

Different patterns of disease activity were observed in relation to the menstrual cycle. Whereas analyses of medical histories revealed that 18 per cent of the female students with severe disease associated aphthous lesions with the onset of the menstrual cycle, 3. 8 per cent of the students with mild disease made this association.[13] Certainly, the relationship of recurrent aphthous ulcerations to endocrinological events in the female (menarche, menstruation, pregnancy, menopause) has been frequently discussed in the past.[6, 15] Examination of incidence associated with menstruation does not confirm our previous data, although both "severe" and "mild" groups of female students displayed considerable disease activity during the premenstrual and menstrual weeks. Because of the divergence of responses between "severe" and "mild" groups, however, it is unlikely that specific endocrine relationships would have primary etiological significance, although the secondary influences probably exert an effect. The definition of these influences must wait until more specific data correlating endocrine changes with oral disease activity are accumulated. Actual determination of ovulation by temperature variations would certainly be more accurate than assuming that a seven-day period would be characteristic. Nevertheless, "premenstrual tension" and the complex psychologic relationships involved with the menstrual cycle, particularly in young adult females, could explain the observations reported.

The mean duration of ulcerations in days and the locations of lesions were constant throughout the twelve months of study and did not vary with seasons, menstrual cycle, or examinations, vacations, and normal school periods. This would suggest that future investigations of this disease in a population can concentrate on the incidence of ulcers rather than duration and location as the primary index of disease activity.

## SUMMARY AND CONCLUSIONS

Following examinations of the oral cavities of 298 students with recurrent aphthous ulcerations attending eight professional schools in the University of Pennsylvania vicinity, monthly diaries recording the location and duration of all intraoral ulcers were collected from 230 students (120 males, 110 females) for periods ranging from three to twelve months. Results indicated large seasonal variations, with increased activity in male and female subjects with both severe and mild disease during the winter and spring months, relative quiescence during the summer, and intermediate levels of activity during the fall months. Students responded to periods of major examinations with increased disease activity, and a decrease in the number of lesions was noted during periods of vacation as compared with normal school periods. In female students different patterns of variation associated with the menstrual cycle were observed when students with severe disease (more than one new lesion monthly) were compared with students with mild disease. The relationship between recurrent aphthous ulcerations and emotional factors has been discussed.

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# ORAL ROENTGENOLOGY

*American Academy of Oral Roentgenology*

*Arthur H. Wuehrmann, Editor*

## HOW HAZARDOUS IS DENTAL ROENTGENOGRAPHY?

*Albert G. Richards, M.S.,* Ann Arbor, Mich.*

THE busy dentist has been bombarded recently with so many conflicting statements regarding the use of x-rays in dentistry that it has become difficult for him to know whether or not the use of his x-ray apparatus presents a hazard to his patients and/or himself. In an effort to answer this question, a search has been made of the dental and medical literature to determine the various clinically proven biologic effects that x-radiation can produce in the human body, the amount of radiation necessary to produce these effects in the various tissues of the body, and the amount of radiation that reaches these same tissues when dental examinations are made with x-rays. From this information, one can determine the degree of hazard that rightfully can be associated with dental x-ray examinations.

### SKIN

The skin of the face is the first tissue exposed in an oral x-ray examination. Excessive exposure of the skin to x-radiation may result in an erythema, or reddening of the skin, which is produced by the dilation of the small blood vessels beneath the skin. The erythema may appear within a few hours following the exposure of the skin.[58] After several days, pigmentation of the skin may replace the erythema. The pigmentation phase may continue for a few weeks to several months.

The dosage of x-rays that will produce the threshold erythema in human skin is reported to lie between 165 and 350 r.[3, 30, 43, 44, 56, 58] It is known that an exposure to fewer r units is sufficient to produce erythema of the skin when radiation of long wavelengths, rather than of short wavelengths, is utilized.[44]

A preponderance of shorter wavelengths can be achieved in an x-ray beam by the application of higher voltages to the x-ray tube or by filtration of the

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*Professor of Dentistry, School of Dentistry, University of Michigan; active member, American Academy of Oral Roentgenology.

x-ray beam. Approximately 50 additional r units of radiation are necessary to produce the threshold erythema for each additional millimeter of aluminum that is added as a filter.[3]

The rate of recovery of human skin from the effects of x-ray irradiation has been found to range between 46 and 77 per cent during the first day,[3, 43, 57] with the percentage of recovery increasing with time.

In 1957 Cade[16] reported that a search of the world's literature uncovered less than 300 reports of cancer induced by radiation. The doses of radiation received by many of the patients were unknown, but when dosages were cited, they were measured in thousands of roentgens. There were no dental patients listed among these patients.

The range of the total dosage of radiation to which the patient's face is exposed during the complete periapical examination is reported to be between 5 and 315 r (measured in air).[51, 59] The wide difference between these two values is due to the different number of exposures, the different amount of filtration, and the different speeds of the films used in making the examinations.

Budowsky and associates[13] have reported that the facial area most heavily exposed to radiation during a complete periapical examination is the skin in the area of the maxillary premolars. When fourteen slow-speed films were exposed to a large unfiltered beam of radiation, this area received between 55.9 and 91.6 r, with 76.9 r as the average exposure. My data[59] indicate that the dose can be reduced to 1.5 r when films of highest speed are exposed to a small, carefully filtered beam of radiation. With only moderately fast films and the same small, carefully filtered beam, the exposure of this area of skin would be closer to 6.5 r.

A comparison of the threshold erythemal dose with the maximum diagnostic exposure of the skin reveals why erythema is never seen in patients, and it also reveals that techniques which use films of the highest speed and small, carefully filtered beams afford the greatest safety to the patient. A similar comparison between the proved carcinogenic dose and the maximum diagnostic exposure of the skin perhaps reveals why there are no reports of oral malignancies attributed to exposures from dental roentgenographic procedures.

Although many millions of dental roentgenographic examinations have been made during the past forty years, no instance of x-ray burns or of facial or oral malignancy resulting from diagnostic dental x-ray procedures has been reported. In the light of this enviable record, plus the fact that far smaller amounts of radiation are being used at present in x-ray examinations of the teeth, no patient need be apprehensive about the possibility of these two effects of radiation occurring following periodic oral x-ray examination.

In 1940 Levin and Behrman[41] reported a case in which pigmentation of the face developed thirty-six hours after an x-ray examination of the patient's jaw. The patient was a 19-year-old girl, the daughter of a white mother and a full-blooded Negro father. The exact roentgen dosage to which the patient's face had been exposed was not known, but it corresponded with that used in the examination of thousands of other patients. The pigmentation appeared without a preceding erythema or other subjective or objective symptoms. It

was thought that the biologic factors, such as race, color, age, and endocrine status, were of much greater importance than the roentgen dosage in the production of the lesion. It was thought also that the pigmentation would disappear within several months without any sequelae. No case similar to this one has ever been reported in the literature.

Some dentists still persist in the hazardous practice of holding film packets in the patient's mouth while the x-ray equipment is operating. This foolish practice has caused some of them to suffer radiation dermatitis of their fingers, others to lose fingers or hands, and still others to lose their lives from radiation-induced cancer.[46, 77] At the Mayo Clinic, a study of 194 patients with chronic x-ray dermatitis of the hands resulting from occupational exposure to x-rays showed that in 31 per cent of the patients the lesions underwent malignant degeneration.[77] In some hands, it took as long as twenty years for the malignant degeneration to develop. There is no record of any patient's fingers suffering damage from radiation because he held film packets within his own mouth. The infrequency of this action by the patient accounts for the absence of damage to his fingers.

### HAIR

Epilation or destruction of hair results from exposure of the skin to radiation in the range from 200 to 600 roentgens.[55, 64, 69, 74] A latent period of about three weeks elapses before the hair is lost. The hair usually will grow back in a few weeks or months. A considerably higher dosage must be given before permanent epilation need be feared.

Two reports were found in the dental literature of over twenty-five years ago in which patients had suffered localized loss of hair.[28, 29, 60] The temporary loss of hair from the scalp occurred after multiple attempts had been made to perform relatively uncommon types of roentgenographic examinations of the head. Loss of hair has never been reported as an aftermath of the conventional types of intraoral x-ray examination.

### EYE

A search of the dental and medical literature has failed to produce a single report of a cataract resulting from exposure of the human eye to x-rays for the purpose of diagnosis. However, the medical literature contains numerous reports of cataracts which resulted from x-ray therapy.[18, 35, 36, 45, 72] In these cases, the eye and its surrounding structures were irradiated with gamma-rays or x-rays in an effort to eliminate a lesion, which usually was malignant, and thereby save the patient's life.

It has been reported that the dose of gamma- or x-radiation required to produce cataracts lies in the range of 200 to 2,000 r.[18, 35, 36, 45, 72] The wide span of this range of doses was explained in part by a study[45] which found that 200 r was the smallest *single* dose that produced a cataract in human eyes, whereas intermediate values of 400 r and 550 r were the smallest total doses that

produced cataracts when the total dose was fractionated (periods of rest interposed between exposures) over periods of between three weeks and three months and over three months, respectively. All patients who received more than 1,150 r to the lens, irrespective of the duration of treatment, developed cataracts.

Eyes of young experimental animals irradiated during the prenatal or early postnatal period show a greater sensitivity to radiation than do the eyes of older animals exposed to the same dose.[32] The lens of the human eye exhibits this same extrasensitivity until the age of 1 year.[45]

The total exposure of eyes in phantom heads has been reported to lie between 3.3 r and 32 r per fourteen-film periapical examination.[4, 38, 71] The use of slow films, no added filters, and x-ray beams of large diameter accounts for the exposure of 32 r reaching the eye. The exposure to 3.3 r was calculated to apply when an x-ray beam of small diameter, filtered externally through 1 mm. of aluminum, was used to expose fast film. The use of additional filtration and extrafast film could have reduced further the irradiation of the eye.

It is highly probable that during a considerable portion of the past years during which dental x-ray examinations have been made, eyes have been exposed repeatedly to doses of radiation that were larger than those just cited from the dental literature.[4, 38, 71] In man a latent period of 2 to 10 years may elapse between the exposure of the eye to ionizing radiation and detection of the resulting lesion.[6, 45] If eyes were damaged years ago by exposure to x-rays in dental offices, ample time now has elapsed for the damage to manifest itself. However, no studies have been reported which establish a correlation between irradiation of the eye from dental sources and damage to the eye. If undetected damage to the eye has actually occurred as a result of the millions of dental x-ray examinations that have been made in the past, it is extremely unlikely to occur again in the future, for present roentgenographic practices employ far smaller doses of filtered radiation administered by beams of radiation that are carefully limited in size.

### TEETH AND JAWS

The lifesaving therapeutic administration of massive doses of x-radiation in the treatment of cancer occurring in proximity to the developing dentition may damage the young teeth and jaws severely. The therapeutic doses of radiation, which are measured frequently in thousands of roentgens, usually cause a retardation in the growth of immature bone and result in the nonformation, dwarfing, premature calcification, and early eruption of permanent teeth.[9, 10, 25, 26, 48] In man no such sequelae to repeated oral examinations with far smaller doses of x-rays has ever been reported.

### BLOOD

The blood-forming organs are very sensitive to irradiation and, fortunately, also have great recuperative powers. In the past it has been considered wise for dentists and other radiation workers to have periodic tests made of their blood to determine whether or not small daily exposures were causing detectable

injury. This opinion has now been challenged by a report that "a large effort at great expense was made by the Atomic Energy Commission during the development of atomic energy to determine if routine hematologic studies would detect low level exposure to radiation. It was the consensus that frequent routine studies on personnel exposed to low levels of radiation have a limited value that does not justify the expense." "Physical control of the environment by radiation monitoring is an effective means of maintaining a safe environment, and nothing is gained by widespread hematologic studies on personnel."[50]

Nolan[51] has reported that significant blood changes have been observed in patients following complete oral examinations with x-rays. On the other hand, Budowsky and his co-workers[14] have reported no significant hematologic changes of clinical importance following complete oral x-ray examinations. The difference in the findings of the two groups of investigators can be explained by the difference in the total exposure that each group of patients received. The patients in the first investigation were exposed to between 115 and 315 r during dental x-ray examinations which consisted of twenty-five to thirty exposures, while the patients in the second investigation were exposed to a total of between 46.0 and 76.9 r during the exposure of fourteen films.

These studies indicate that dental roentgenographic examinations can be performed without producing hematologic changes, provided that the total dosage is kept reasonably small.

## LEUKEMIA

Leukemia is a fatal disease of the blood-producing organs, such as the lymph glands and the bone marrow. During the period from 1921 through 1955, the mortality attributed to leukemia rose steadily. Since about 1940, however, there has been a perceptible decline in the rate of increase of incidence of leukemia in the white race, except in persons 75 years of age and over. For the entire population, the per cent increase dropped from 64 per cent between 1930 and 1940 to 43 per cent between 1940 and 1950. If the rate of increase continues to decline in the future, the leukemia death rates for all ages will become stabilized or perhaps even decline.[34] From these trends, it appears that, although both the exposure of the population to ionizing radiation and the incidence of leukemia have increased since 1940, the rate of increase in the incidence of leukemia has dropped while the exposure continues to rise. In other words, while the rate has dropped, the actual incidence has increased but not to the same degree as in previous decades. Between 1950 and 1955 the incidence of leukemia among white persons of all ages was 77.1 and 53.2 per million for males and females, respectively.[34]

The etiology of leukemia is not known completely, but there is evidence that ionizing radiation, carcinogenic chemicals, viruses, and heredity may be leukemogenic agents.[24] There is considerable evidence which connects ionizing radiation with the induction of leukemia in man, but only about 15 per cent of the cases of leukemia can reasonably be ascribed to radiation.[76]

The evidence relating to radiation-induced leukemia is based on reports of an increase in the incidence of leukemia among the following groups who have experienced extensive irradiation: (1) medical radiologists,[27, 34, 37, 54, 75] (2) survivors of the atomic bombings in Japan,[47, 50, 73] (3) patients treated for spondylitis,[20-22] (4) children treated for enlargement of the thymus gland,[66] and, (5) children of women who had abdominal radiography while pregnant.[68]

In the third and fourth groups the irradiation was limited to specific portions of the body and hence would be considered partial-body exposure, whereas the other groups received whole-body exposures. In all these reports, the major portion of the red marrow in the chest and spine of the subject was irradiated. In the first group, the total chronic exposure is estimated at about 2,000 r delivered over many years.[7] In the second and fourth groups the acute exposure probably exceeded 125 r. In only two of the cases in the third group were the mean doses to the bone marrow less than 470 r, while in the fifth group the acute fetal exposure probably was about 2.5 r.[52]

Available data are insufficient to support any conclusion concerning the shape of the radiation-dose–leukemia-response curve, particularly in the low-dose region. Statements attributing great accuracy to radiation-induced leukemia rates in human populations are open to serious question, particularly those which suggest a strictly linear dose-response relationship.[11, 40]

Among the survivors of the atomic bombings in Japan and among the patients given x-ray therapy for spondylitis, a latent period of three to six years passed between irradiation and diagnosis of leukemia.[23]

The doses of radiation received by the sternum, ribs, and spinal column during oral x-ray examinations are not known, but they are believed to be small since the chest is not exposed directly during dental examinations. Also the x-ray beam exposes relatively small areas, thus producing partial-body exposures. Dental exposures also are fractionated widely in time. The factors of fractionation and partial-body exposures tend to diminish greatly any effect which the radiation may produce in the somatic tissues of the body. The amount of red marrow tissue that is irradiated during oral x-ray examinations is but a very small fraction of the patient's total red marrow; hence, the hazard of leukemia induced by the small contribution from dental x-ray procedures to the irradiation of the whole body may be very slight.

A complete periapical x-ray examination of a pregnant woman may expose the fetus to 0.8 mr.[67] The use of a small, properly filtered beam of x-rays and films of the highest speed can further reduce the x-ray exposure of the fetus to such a minute amount that the possibility of the fetus' being damaged is negligible.

During 1956 the Bureau of Economic Research and Statistics of the American Dental Association[15] issued a report listing essentially the same percentage of deaths from leukemia among dentists as has been reported for white males in the United States.[34] (See Editor's note.*)

*_Editor's note:_ The facts presented by Mr. Richards are correct. However, it is important to emphasize that the persons reported in the American Dental Association's study[15] were predominantly in the older age group and hence may not have used x-radiation extensively. No effort was made to correlate the degree of radiation usage with the cause of death. Similar statistics for the group graduating from dental school after 1935 could show different results. The number of dentists using radiation has increased markedly during the last twenty-five years.

Increasingly large numbers of patients have undergone repeated dental roentgenographic examinations in the past. The periods of time that have elapsed since then exceed the latent period for the occurrence of leukemia. Yet, in the presence of an increasing exposure of the population to x-radiation for the purpose of dental diagnosis, the rate of increase of the incidence of leukemia has declined steadily for the past twenty years. Apparently, then, the occurrence of leukemia as the result of the x-radiation used in dentistry need not be feared greatly by either the dental patient or the dentist.

### THYROID GLAND

Cancer of the thyroid gland has been reported in a few instances among many children who were given x-ray therapy (200 to 800 r in air) directed at the head, the neck, and the region of the thymus gland. This suggests that the child's thyroid gland is especially sensitive to irradiation.[50, 61, 65] Doses of less than 180 r to the thoraxes of children have been found not to produce malignant tumors in the thyroid glands of these patients.[61] The adult's thyroid gland must be less sensitive to irradiation than the child's for very few cases have been reported of thyroid cancer developing in patients who had received thyroid gland irradiation during adulthood.[33]

Calculations made with the data provided by Baily[1] and Bjärngard[2] relating to the exposure of the thyroid indicate that less than 1 per cent of the total dose received by the face during a fourteen-film periapical examination is effective at the thyroid gland. The exposure of the thyroid gland would be larger, of course, if x-ray beams of large diameter were used.

No studies made in an attempt to prove or disprove an association between dental exposure of the thyroid and malignancy of the gland have been reported.

If the small exposure of the thyroid gland to radiation during dental x-ray examinations has caused damage to the gland in the past (and this has never been proved), it is far less likely to occur in the future because of the greatly reduced amounts of radiation that are currently being used in dentistry.

### LONGEVITY

It has been demonstrated that the life span of a small laboratory animal can be shortened by exposure of the animal's entire body to x-radiation. Some data[39] indicate that the reduction of life span is proportional to the total dose. However, some careful longevity studies with mice have shown an increase in the average life span at low chronic doses; others have shown that shortening of life actually is not proportional to dose.[5, 12] Decreased longevity of American radiologists has been reported by one observer[75] and denied by others.[42, 63, 70] The life expectancy of British radiologists[19] was found to be similar to that of other men of comparable social status and has not been altered substantially by occupational exposure to radiation.

Experiments with mice exposed to 300 to 1,800 r have shown for a given tissue dose that partial-body exposure was much less effective than whole-body

exposure in the shortening of life.[39] Extrapolation of the data for partial-body exposure suggests that at still lower doses (for example, exposure at 100 r) the effectiveness per unit-dose may be so reduced as to be negligible. The doses tested in this study on mice are hundreds to thousands of times greater than those used in human roentgenographic diagnosis. Moreover, the total dose accumulated from repeated diagnostic exposures is fractionated and, therefore, presumably of diminished effectiveness.[39]

A statistical study of the longevity of dentists indicates that dentists live almost three years longer than white nondentists.[15] The result of this study must not be construed to indicate that the dentist's small daily occupational exposure to x-radiation has lengthened his life span; instead, these data indicate that apparently the dentist's life expectancy has not suffered because of his slight occupational exposure to radiation.

In the light of the evidence presented, neither the dental patient nor the dentist needs to worry seriously that his contact with x-ray radiation in the dental office will have any effect upon his life span. The evidence, however, is by no means complete.

## STERILITY

Sterility results from the destruction by x-radiation of the gonadal tissues which produce mature sperm and ova. Whether the tissue destruction and the subsequent sterility are temporary or permanent depends on the dose of radiation received by the gonads. It has been reported that an estimated 400 r in a single acute dose to the male gonads will produce permanent sterilization, whereas the sterilizing dose in the female may be more than 625 r (effective at the ovaries).[8, 36]

No data are available regarding the amount of chronic exposure that will produce either temporary or permanent sterility in human beings. Presumably, because of the repair that occurs in gonadal tissues, a greater total dose of radiation from chronic exposure than from an acute exposure would be necessary to produce sterility.

In contrast to the dose that will produce permanent sterility, the exposure of the male gonads resulting from complete dental x-ray examinations has been reported to range from 0.00043 r to 0.07 r.[17, 31, 59, 67] The probability that sterility might result from the amount of irradiation that the gonads receive during a complete oral x-ray examination is therefore extremely remote. Even the annual repetition of the x-ray examination would not significantly increase the possible occurrence of permanent sterility in the male patient. Because of the shielded location of the ovaries, the gonads of the female patient receive about 18 to 44 per cent as much radiation from oral x-ray examinations as do the male gonads.[2, 67]

If a dentist is conscientious about limiting the exposure of his own body and gonads to no more than the current official permissible exposure of 5 r per year, he never need fear that sterility will occur because of his occupational exposure.

GENETIC EFFECTS

The genetic effects of ionizing radiation on man are largely theoretical, since little has been proved regarding them. This lack of proof stems from the great difficulties associated with obtaining quantitative evidence. Much of the knowledge regarding mutations induced by ionizing radiation has been obtained from experimentation with plants, insects, and mice.

One of the basic tenets of radiation genetics has been that the mutagenic effect of ionizing radiation is a direct function of total dose and independent of dose rate. This conclusion originally was based upon experiments in which the spermatozoa of the fruit fly, *Drosophila*, was irradiated. Russell and co-workers[62] have confirmed with mice that the mutagenic effect of ionizing radiation on the spermatozoa is a direct function of the total dose and independent of the dose rate.

Russell reasoned that it was the spermatogonia and oocytes, and not the spermatozoa and ova, which were present in the mammal from birth, and that the experience of these cells with radiation, rather than that of the spermatozoa and ova, was of greatest genetic importance. Russell's research indicates that less genetic damage is done to the spermatogonia and oocytes at lower rates of exposure than at higher rates. For chronic dose rates (10 to 90 r per week), the genetic effect on the male mouse is about one fourth and on the female mouse about one seventh of the effect caused by acute doses of 80 to 90 r per minute. Recently, it has been proved that the lower mutagenic effectiveness of chronic radiation over acute radiation holds true also in the *Drosophila*.[53] From the results of Russell's investigation, it appears that the genetic hazards, at least under some radiation conditions, may not be as great as those estimated from the mutation rates obtained with acute irradiation. The discovery of the reduced mutagenicity of chronic irradiation therefore has introduced into the field of radiology a factor of safety which previously was not known to exist.

The upper limit of the rate of chronic irradiation reported by Russell is 90 r per week or 0.15 mr per second. It is a matter of record that, under optimal conditions, the male gonads are irradiated at the rate of 0.03 to 0.34 mr per second when 60 to 65 KVP are used in making dental x-ray examinations[2, 67] and 0.31 mr per second when 90 KVP are used.[59] The dental irradiation of the female gonads has been reported to be at the rate of 0.01 to 0.06 mr per second when oral x-ray examinations are made with 60 to 65 KVP.[2, 67] Filtration, collimation, voltage, current, focal-skin distance, angulation, and length of torso are the physical factors which control the rate of exposure of the gonads. From these data regarding dose rates, it appears that the rates at which the gonads are irradiated during dental x-ray examinations lie in the approximate range which Russell found to be less hazardous than was thought previously.

The National Academy of Sciences[49] has recommended that "the average exposure of the population's reproductive cells to radiation above the natural background should be limited to 10 roentgens from conception to age 30."

The total amount of radiation reaching the gonads during a complete periapical examination of the mouth with fourteen exposures is very small,[17, 59] between 0.00043 r and 0.07 r. It is well to remember that only the radiation that reaches the gonads can be considered harmful from a genetic standpoint, and then only in persons who are under or within the child-bearing age.

To make certain that the dental contribution to the total gonadal exposure of the population is very small, each dentist should consider carefully whether he can reduce the amount of radiation that he uses for each dental x-ray examination.

## CONCLUSIONS

1. A survey of the dental and medical literature of the past forty years has failed to disclose any clinical evidence that would seriously condemn the practice of dental roentgenography when used in an intelligent manner, as being unsafe for either the patient or the dentist.

2. The few documented incidents of untoward biologic effects that have occurred in the past when dental patients were exposed to larger amounts of x-radiation are less likely to be repeated among the patients of today because of the far smaller amounts of x-radiation that presently can be employed.

3. There still are areas in which there is no clinical evidence that either proves or disproves that a correlation exists between a given dental exposure to radiation and a specific biologic effect. In view of this uncertainty, it is prudent to keep to a minimum the amount of ionizing radiation that a person receives from all artificial sources of radiation; therefore, each dentist should review his current roentgenographic procedures to see if his examinations could be made with less radiation.

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## ROENTGENO-QUESTIONS

*I have a William Meyer x-ray machine, Model CA3, and I would like to equip it with an electronic timer. However, my serviceman tells me that, because of the short exposures, the timer would not allow the head to heat up properly and it would greatly shorten the life of the unit. I do not know if this is a correct explanation or if it is true, but I would appreciate your opinion.*

*Editor's Note:* The answer to the above question was not immediately available. An alternative answer was given, and an attempt was made to obtain more specific information. Because both answers are of value, they are reproduced. The second reply mentions a particular product. Ordinarily, product names are deleted but, as stated in the reply, specific reference is necessary. Appreciation is expressed to Mr. Walter S. Strauss for his cooperative assistance.

*Answer 1.* Unfortunately, I am not well informed about the Meyer x-ray machines, but I can readily understand the remarks of your serviceman since the use of short time exposures necessitates a preheated tube filament. As an alternative, let me suggest the use of an extension cone. (Doubling your present target-to-film distance would necessitate an increase in exposure time by a factor of 4.) If you are contemplating the use of fast film (for example, ultra-speed film versus radiatized film), the exposure times would not be changed with the use of a long cone since the speed of the ultra-speed film is approximately four times faster than that of the radiatized film. Another alternative is to reduce the tube filament current. Most dental machines operate on 10 Ma. By inserting a resistor in the tube filament circuit, the Ma. can be reduced as desired. Since exposure is a matter of Ma. $\times$ seconds, a reduction in Ma. permits an increased number of seconds of exposure.

*Answer 2.* Two basic methods for preheating x-ray tubes are used in most machines. The first method energizes the x-ray tube filament as soon as the main line switch of the machine is turned on. The Universal Model U2550, the Fisher Models MD, SD, and WD, the Meyer Model C, and a few of the

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The American Academy of Oral Roentgenology solicits from the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY any questions which deal with technical or interpretive aspects of oral roentgenology. These questions should be sent directly to the editor of this section, Dr. Arthur H. Wuehrmann, School of Dentistry, University of Alabama Medical Center, Birmingham, Alabama; the editor will distribute them for reply to a suitable group within the Academy. Questions, with the answers, will be published at as early a date as possible in "Roentgeno-Questions." Questions, especially those of an interpretive nature, should ordinarily be accompanied by good-quality roentgenograms and a pertinent history. The names of the questioner and the respondent will not be published.

Weber machines are in this category. Unfortunately, we have no circuit diagrams availabile for the Meyer x-ray machine mentioned in the question, because this company is no longer in business. We have checked only the one particular Meyer machine mentioned previously and found that the tube filament is turned on when the line switch is on so that the tube is actually preheated before the exposure is started.

In the second method the tube is preheated when the button on the mechanical timer is depressed. The Ritter B and older Dual-X, the General Electric ECDX, and most Weber machines are in this category. Partial depression of the mechanical timer button completes the preheat circuit and full travel starts the exposure.

The Hanau electronic timer has incorporated an automatic one-half-second delay to permit the tube filament to preheat before the actual exposure. The electrical sequence is the same with the Hanau timer as with the mechanical timer, except that all switching is electronically controlled. Thus, the preheat period is electrically operated rather than hand operated as was previously the case with the mechanical timer. This also assures the operator that the tube is capable of emitting full radiation during the entire exposure, thus avoiding the undesirable necessity for adding preheat time to the actual desired exposure interval.

The newer Ritter Dual-X contains a micro-switch-operated timer hanger. As soon as the timer is removed from the hanger, the filament of the x-ray tube is energized. The Hanau timer utilizes the same electrical circuit for preheating the tube, except that the micro switch on the machine is replaced by a relay which is electrically timed to provide one-half-second preheat.

It is therefore our opinion, as evident from the above, that the Hanau timer does not alter the function of the x-ray machine or subtract from the life of the machine or any of its parts. The timer does provide precise timing of exposures utilizing existing circuits already present in each machine.

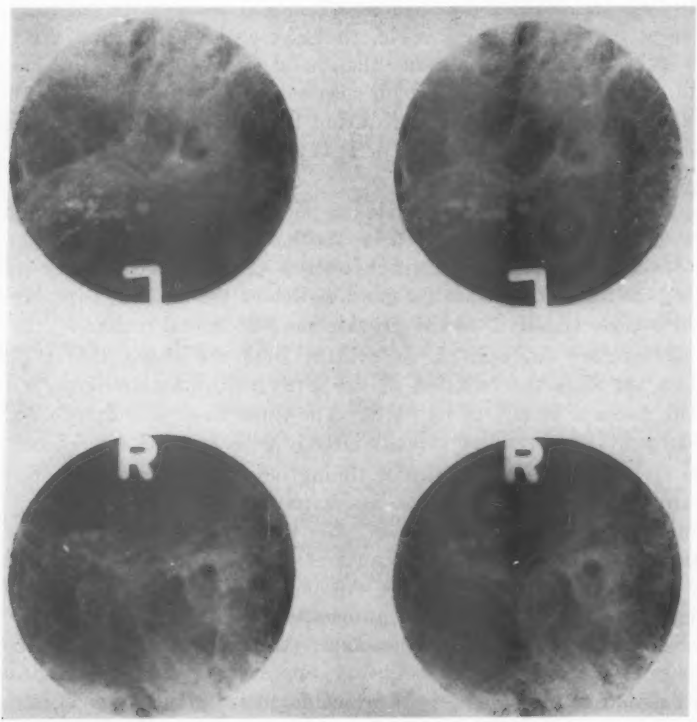
The advantages of electronically timing older x-ray machines are readily appreciated when high-speed films are exposed at only three-tenths second. This minimizes the possibilities of cumulative radiation absorption by the dentist, the patient, and the personnel. Because of precise electronic timing, the consistency of roentgenographic quality will improve significantly. As a further convenience, electronic timers reset automatically for the next exposure at the same selected speed. On the Hanau timer, the full scale of dental x-ray requirements is covered with twenty-four exposure times, ranging from one-thirtieth second to one second in fourteen increments and from one second to six seconds in ten one-half-second increments.

We readily understand your desire not to refer specifically to Hanau in any written material which may appear in your journal. We do, however, wish to call to your attention the fact that other electronic timers on the market do not necessarily function like the Hanau timer. It is therefore impossible for us to generalize on the subject without specifically referring to the Hanau timer.

## ROENTGENO-ODDITIES

### Temporomandibular Joint Roentgenography

THE roentgenograms of the temporomandibular joint which are shown here demonstrate a kind of "buckshot" appearance above and anterior to the articular fossa. At first this was thought to be an artifact, but additional films substantiate the findings.




Each month this section will bring to the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY one or more roentgenograms which demonstrate unusual, unexpected, rare, or bizarre roentgenographic changes. These roentgenograms will be accompanied by an explanation or by words of inquiry regarding the particular change. All films used in this section will be accepted with the understanding that 2 by 2 or 3¼ by 4 inch slides or glossy photographs of the films will be made available to interested readers through the editor of the American Academy of Oral Roentgenology at the individual's expense. The cost will involve only the photographer's charges. Each slide or print will give credit to the donor. Interesting roentgenograms are solicited from all sources. Please be certain to identify your films properly so that they can be returned. All material for publication should be submitted to Dr. Arthur H. Wuehrmann, University of Alabama School of Dentistry, Medical Center, Birmingham, Alabama.

A more detailed history revealed that a myelogram had been taken eight months earlier. It was finally decided, in consultation with the patient's neurologist, that Pantopaque had migrated into the basal cisterns of the cerebrospinal circulation. The physician stated that the findings were of no importance and that probably within ten years the radiopaque material would disappear from the cerebrospinal circulation.

*Robert I. Litwin*
*Brookline, Mass.*

# ORAL PATHOLOGY

*American Academy of Oral Pathology*

*Donald Kerr, Editor*

## MALIGNANT PERIPHERAL NERVE TUMORS OF THE ORAL CAVITY

### Review of the Literature and Report of a Case

*Duane T. DeVore, D.D.S.,* and Charles A. Waldron, D.D.S., M.S.D.,***
*Atlanta, Ga.*

INTRODUCTION

IN THE study of tumors of the peripheral nerves there is a great deal of confusion as to terminology, origin, and general classification of both the benign and the malignant forms. According to Boyd,[9] "The histology of the nerve trunk (tumor) seems quite simple and well understood if one reads but a single authoritative paper on the subject, but should one read several, it will take on more and more complexity." It would be expected that the newer methods of experimental technique and staining should have brought about some degree of clarification; however, they seem to have only added to the confusion.

Prior to a discussion of any nerve sheath tumor, a short review of the structure of the normal nerve sheath is in order. The medullated nerve fiber is covered by a thin layer of myelin, which is thought to be the insulation of the nerve. Around the nerve fiber, either myelinated or unmyelinated, there is a supporting layer called the neurilemma or the sheath of Schwann. This sheath is of ectodermal origin. At regular intervals along the nerve fiber this sheath is interrupted at the nodes of Ranvier. A group of these fibers are bound together by a covering known as the perineurium, and a group of these bundles are then bound together by the epineurium. This entire group is the nerve proper.[19]

The patient was treated at the Veteran's Administration Hospital, Atlanta, Georgia, while the senior author was a resident in oral surgery. The authors wish to thank Doctor Simon Krantz, Chief of Radiology.

*Associate in Pathology, Emory University School of Dentistry. Present address: 811 E. 66th St., Savannah, Georgia. Formerly American Cancer Society Fellow in Oral Pathology, Emory University School of Dentistry.

**Professor of Oral Pathology, Emory University School of Dentistry.

A review of the dental literature since 1839 and the medical literature for the past fifty years reveals a multitude of terms for what are apparently similar lesions. Because of the lack of adequate microscopic descriptions in the earlier literature, one can only surmise, from the history, clinical course, and location, the exact type of tumor under discussion. Some of the various terms used for the benign tumor are neurofibroma, perineural fibroblastoma, neurilemmoma, neurilemoma, neurinoma, Schwannoma, peripheral glioma, fibroma of the nerve sheath, neuroma, cerebellopontine angle tumor, tumor of the nervus acousticus, glioma of the acoustic nerve, endothelioma, benign plexiform neurilemoma, Von Recklinghausen's disease, neuroblastoma, and neurogenic fibroma. Some terms used for the malignant form are neurogenic sarcoma, neurogenous sarcoma, fibrosarcoma, neurofibrosarcoma, malignant Schwannoma, malignant neurinoma, malignant neurilemmoma, fibromyxosarcoma of nerve, Schwannosarcoma, malignant peripheral glioma, myxosarcoma of nerve sheath, neurilemmosarcoma, and sarcoma of the peripheral nerve.

More recent literature indicates that there are essentially two schools of thought as to the histogenesis of these tumors. Foot[28] states: "As the embryonal origin of both groups has not yet been firmly established, the reason for this controversy is evident; that which seems incontrovertible to one camp is lightly brushed aside by the other as being of no consequence." The first is the group that follows the views of Penfield and Mallory in believing that these tumors are composed of modified fibroblasts. The second, and by far the most popular, concept is that set forth by Masson and Stout, who believe that these tumors are of Schwannian origin. It is noted, however, that Penfield described a "malignant Schwannoma," although he stated that he could not explain this tumor.[28] Both groups believe that the term "peripheral glioma" is a correct designation, but since Penfield and his associates used the term first, the term "Schwannoma" was adopted by Masson.[46, 55] Verocay,[69] in 1910, suggested the term "neurinoma" for these peripheral nerve tumors. This designation, however, implies that the tumor is of nerve-cell origin, and it is seldom used today.

In 1920 Antoni published an extensive monograph in which he described two histologic variants of peripheral nerve tumors. His criteria for type A and type B tissue are widely used today. Type A tissue is characterized by an orderly arrangement of elongated spindle cells which are usually parallel to each other. There is also a parallel arrangement of intercellular fibers to the long axis of the nuclei. This lining-up of parallel rows of cell nuclei is called palisading. The nuclei may produce a pattern of whorling, called Verocay bodies. In type B tissue there is no arrangement of the fibers or cells, as there is in type A. There is a loose arrangement of variably shaped cells. Intercellular vacuoles and microcystic degeneration are sometimes seen. The intercellular fibers are composed of reticulum, and the collagen bands are less numerous. In 1932 Penfield,[55] in an attempt to standardize terminology, introduced the term "perineural fibroblastoma." He felt that these tumors do not contain nerve fibers and that the cell type is not the Schwann cell but connective tissue, perineural, and endoneurial cells. Also in 1932, Masson[46] came out strongly with evidence to support the Schwannian theory. He pointedly stated at the

opening of his paper that "the following pages are addressed to the minority of pathologists who still refuse to admit that the fundamental constituent of the encapsulated tumors of the peripheral nerve is the Schwannian syncytium, a neuroectodermal structure, and not the mesodermal fibroblast." He showed experimentally that when a trichrome stain was used the Schwann cells stained red, whereas the fibrous elements took up a green or blue (collagenophil) stain. Stout,[64] in 1935, agreed and added the term "neurilemoma," meaning nerve sheath tumor. On the other hand, Tarlov[68] stated in 1940: "Direct staining evidence by a technic that brings out normal and proliferating Schwann cells excludes the Schwann cell as the type cell of the common encapsulated tumor of peripheral nerves." Nineteen pages later in the same journal, Murray, Stout, and Bradley[50] stated: "We have offered evidence, obtained by means of tissue culture, that the specific nerve sheath tumor or neurilemoma is of Schwannian origin." Since 1940 numerous papers have presented the pros and cons of the Schwannian theory of origin of both the benign and the malignant nerve sheath tumors. There is no need here to go into any more detail concerning the remainder of these articles, as no theory is conclusive at this time.

The classification and terminology advocated by Stout[67] appears to be widely used by pathologists today. This classification includes the following:

1. Neurofibroma
2. Neurilemmoma (Schwannoma)
3. Malignant Schwannoma

Malignant neoplasms arising from peripheral nerves have also been the subject of considerable controversy with regard to histogenesis and proper designation. In 1927 Quick and Cutler,[56] along with Ewing, made a study of seventy-five cases at Memorial Hospital. They felt that most subcutaneous intermuscular tumors which were commonly designated as fibrosarcoma, spindle-cell sarcoma, and fascial sarcoma are actually of neurogenic origin. They classified these tumors into three groups:

> Grade I. Tumors of low malignancy. These are relatively acellular, fibrous tumors composed of large spindle cells.
>
> Grade II. Tumors of intermediate malignancy. They are composed of large spindle cells arranged in bundles with whorl formation. There is very little intercellular substance, and the cells are predominant.
>
> Grade III. Highly malignant tumors. These are very cellular and are composed of small spindle cells arranged in whorls. They may be composed of polyhedral cells in a loose network.

The diagnosis of neurofibrosarcoma was apparently very popular and even "epidemic" twenty to thirty years ago.[1] A clearer delineation in recent years of the various malignant soft-tissue tumors has clarified some of this problem, and most pathologists today agree that malignant tumors of peripheral nerve are rare neoplasms.[66]

Stout,[65] in 1935, divided malignant peripheral nerve tumors into two groups. The first group consisted of tumors which had the morphologic characteristics of malignant fibroblastic tumors. These included malignant degeneration of

The neurofibromas of Von Recklinghausen's disease, which has been estimated to occur in 10 to 15 per cent of cases. The second group consisted of tumors which were more of a neuro-entodermal structure. Stout claimed that both types developed from the nerve sheath, although the first type was encountered more frequently. Most authors believe that the encapsulated specific nerve sheath tumor (neurilemmoma, Schwannoma) is completely benign and does not undergo malignant transformation. Malignant nerve sheath tumors are believed to arise either from a pre-existing neurofibroma of Von Recklinghausen's disease or de novo from elements of the nerve sheath. [3, 56, 63, 64, 65, 67] Some authors, however, believe that specific nerve sheath tumors may, on rare occasions, undergo malignant change.[10, 15, 71]

A number of cases of benign nerve sheath tumors of the oral cavity have been reported.[3, 7, 8, 10, 12, 15, 16, 19, 21, 22, 32, 33, 37, 39, 45, 57, 60, 62, 64, 73] A partial listing of these cases shows involvement of the tongue (five cases), submaxillary region (four cases), body of the mandible (three cases), inferior alveolar nerve (three cases), buccal mucosa (three cases), soft palate (two cases), hard palate (two cases), labial sulcus (two cases), and retromolar area (one case). Multiple neurofibromatosis involving the lips, tongue, and gingivae has also been reported.

An exhaustive review of the literature revealed two cases of malignant neurogenous tumors involving the maxilla and seven involving the mandible and/or the inferior alveolar nerve. Cutler and Gross[19] describe the case of a 36-year-old white woman who had pain and swelling of seven years' duration in the temporal region. Two operations were performed and on both occasions the lesion was diagnosed as benign. The lesion recurred again and involved the palate. At the third operation a diagnosis of neurofibrosarcoma was made. Millard and Busser[47] also reported a malignant neurogenous tumor of the maxilla which caused the death of a 42-year-old woman.

The mandibular cases show several distinct clinical patterns. Bell[5] reported an exophytic growth in the mandibular premolar-molar region of a 65-year-old woman. This was removed and a microscopic diagnosis of neurogenic sarcoma was made. No follow-up information was given on this case. Wilson and Walsh[72] reported the case of a 2-year-old girl who appeared to have a large cystic lesion of the mandible. At operation the area was found to contain a solid tumor which appeared to arise lingual to the mandible and was invading the bone. The initial impression was neurogenic fibroma, but follow-up indicated that the lesion was in reality a neurogenic sarcoma. DeLarue[23, 24] also reported a malignant Schwannoma of the mandible in a 30-year-old man. This lesion presented radiographic features suggesting a cystic lesion at the apex of a lower premolar.

The remaining four cases bear a striking similarity in their history and clinical features.

Lewis and Hart[41] reported the case of a 41-year-old man who noted a hard, nonulcerated nodule on his lower lip. This lesion appeared to be encapsulated. The lesion was enucleated but recurred fifteen times over a period of three years. A diagnosis of neurofibroma was made on each occasion. Three years after the first operation the mandible and submaxillary gland were resected. At this time

it was noted that the inferior alveolar nerve and canal were greatly enlarged and that the tumor extended to the base of the skull. The lesion was cauterized at the point where it entered the skull. Microscopically, the tumor was diagnosed as a "sarcoma." The patient died the next year with cerebellar symptoms. There was no evidence of Von Recklinghausen's disease in this case.

Wise and Asbury[74] reported a case in which a 31-year-old white man had pain and tingling in his lower lip. Seven months prior to his being seen by the authors, a V-excision of the lip lesion was performed and a diagnosis of sarcoma was rendered. Roentgenograms taken six months postoperatively showed an enlarged mental foramen. A mass was noted at the mental foramen. The mandible was resected and the nerve was removed at the base of the skull. A diagnosis of "sarcoma" was again made. A craniotomy was then performed, and microscopic sections of the gasserian ganglion revealed a tumor in the mandibular branch. Six months later a recurrence in the mental region was excised. The patient was apparently free of disease during the next five months, at which time the case was reported.

In a case reported by Cuneo and Rand,[18] a 37-year-old white man had numbness of the left lower lip which gradually spread through the mandible to the tragus of the ear and over the cheek. He also had pain in the anterior two-thirds of the tongue. Roentgenograms revealed an enlargement of the inferior dental canal and mental foramen. There was also an enlargement of the left foramen ovale. A biopsy of the mental foramen revealed only marked inflammation. At operation, the tumor was excised at the foramen ovale and cauterized. The microscopic diagnosis was made by two men; one said that it was probably a neurofibroma, and the other stated that it was a malignant Schwannoma. A three months' postoperative follow-up was reported. At this time the patient was free of disease.

Economou, Southwick, and Slaughter[25] reported the case of a 56-year-old white man who had injured his lip as a child; a lump had been present ever since. In 1949 the lesion enlarged and became ulcerated. It was excised and reported as a benign lesion. It ulcerated again one month later. In 1951 a mass was seen on the midportion of the lower lip. It was excised and reported as a "neurofibrosarcoma." The lesion recurred eighteen months later and was again widely excised. Five months later a lesion appeared over the mental foramen. The mandible was subsequently removed and the nerve was excised to the foramen ovale. The nerve at this point was negative for tumor. However, there was a tumor alongside the nerve. There was no evidence of recurrence at the time the case was reported in the literature.

At this point a new case currently under observation will be presented.

H. T., a 65-year-old white farmer, was admitted to the hospital in March, 1959, for removal of bilateral senile cataracts. The past history revealed that the patient had diabetes mellitus for which he had been receiving treatment from his physician. In December, 1956, the patient noted a small ulcer on the right lower lip in the region where he habitually held his cigarette. The lesion was removed elsewhere by V excision and was reported as squamous-cell carcinoma. Upon questioning in 1959, the patient stated that for the past two years there had been a "numbness" in his right lower lip, but he attributed it to the excision of the lesion. He also stated that recently his lower dentures had not fitted properly. Also, within

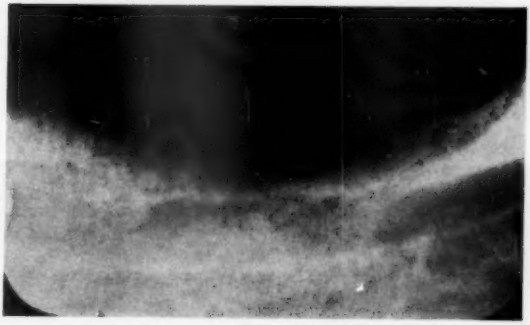


Fig. 1.—Dental film taken at time of admission showing a smooth enlargement of the right mandibular canal.

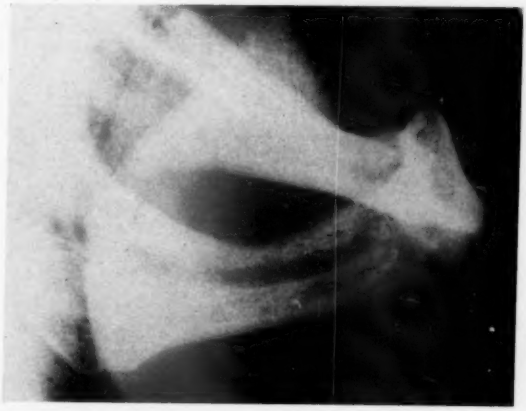


Fig. 2.—Right lateral jaw roentgenogram taken at time of admission.

Fig. 3.—Left lateral jaw roentgenogram taken at time of admission demonstrating a more normal size of mandibular canal.

the past few months the "numbness" had begun to extend further along the body of the mandible and the lateral border of the tongue, and the patient also had experienced some mild shooting pains of the right upper lip. Examination revealed a nontender, nonulcerated, firm 3 by 1 cm. mass over the right mental foramen area. The remainder of the oral examination revealed edentulous ridges in an otherwise apparently normal oral cavity. The

Fig. 4.

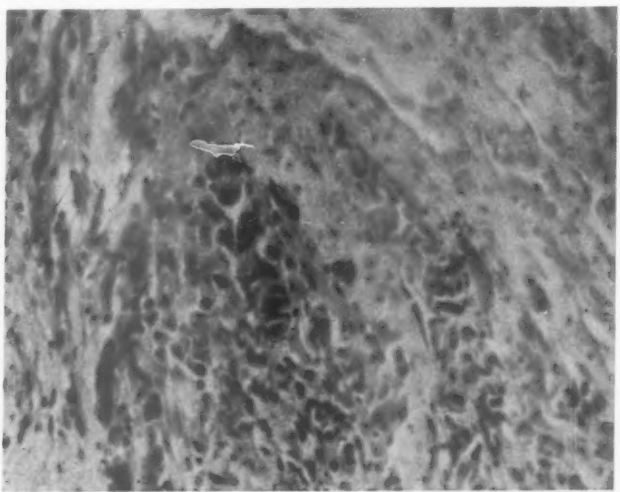


Fig. 5.

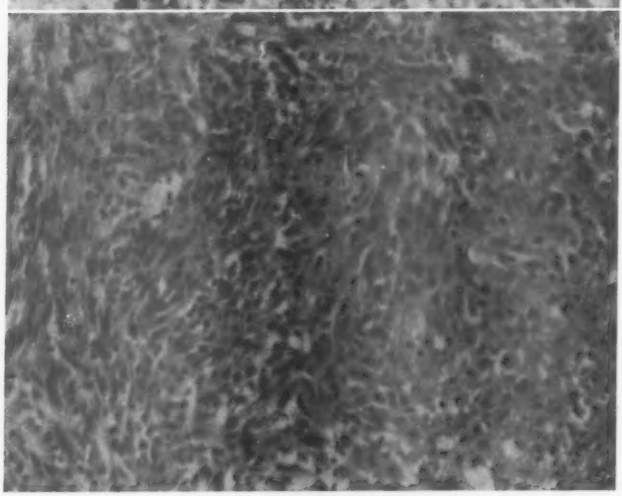


Fig. 4.—Low-power photomicrograph of biopsy specimen from the right mental foramen area showing neoplastic involvement of nerve bundle.

Fig. 5.—Another area from biopsy specimen showing a spindle-celled neoplasm with a fasciculated pattern.

physical examination revealed a mild hypertension, but otherwise the findings were essentially normal. Fasting blood sugar and glucose-tolerance tests were positive for diabetes mellitus. Roentgenograms revealed a marked enlargement of the canal of the inferior alveolar nerve

on the right side (Figs. 1, 2, and 3). Basilar skull views showed a clouding over the right foramen ovale area, with possible early destruction. An exploratory biopsy of the right mental foramen was performed on March 23, 1959. When the mass was entered, it appeared to be growing out of the mental foramen and sending many fingerlike projections into the surrounding tissue. The mass was firm, slightly yellow in color, and of uniform consistency. An adequate section was removed for microscopic study, and the area was closed with silk suture.

*Pathologist's Report.—*

*Gross:* The specimen consisted of four pieces of tissue, the largest being an irregular club-shaped mass measuring 3.0 by 1.0 cm. at its widest point.

*Microscopic:* These sections showed a neoplasm composed of large, hyperchromatic spindle- and ovoid-shaped cells which were arranged in a fasciculated pattern. In some areas the cells suggested a palisade arrangement, with alternating cellular and acellular areas. Occasional mitotic figures were observed. Some of these islands of tumor cells appeared to be arising directly in large nerve bundles, while others simulated the appearance of a nerve bundle. A number of apparently uninvolved nerve bundles were also observed.

*Diagnosis:* Malignant Schwannoma (Figs. 4 and 5). The sections were reviewed by several other consultants, all of whom concurred with the diagnosis.

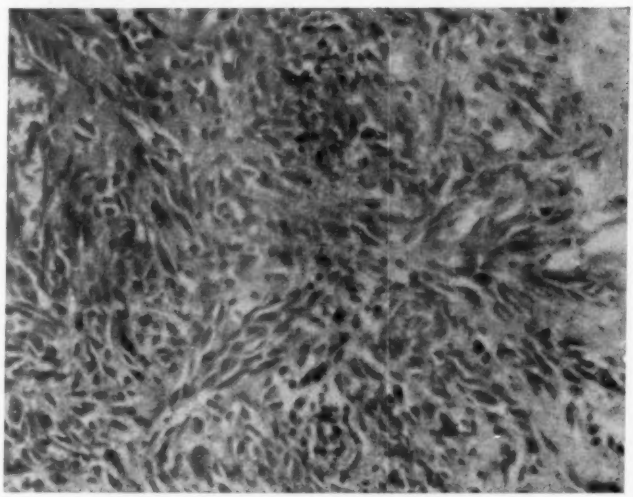


Fig. 6.—Photomicrograph of section of lip excised in 1956. This shows a spindle-celled neoplasm with a fasciculated pattern.

Slides of the specimen excised in 1956 were obtained and reviewed. A revised diagnosis of malignant Schwannoma of the right lower lip was made (Fig. 6). Within one week after we first saw the patient, he began to notice paresthesia in his right upper lip which extended as shooting pains to the right infraorbital region. After consultation, it was decided that, because of the patient's age, general condition, and probable extension of the tumor, the patient should be given radiation therapy. X-ray therapy was started on April 10, 1959; the patient received a total dose of 2,850 r to the right mandible and 2,850 r to the right preauricular region. The patient was again examined three months later, and the mass over the mental foramen was no longer visible or palpable. The patient still had the paresthesia in the previously noted areas. Six months after therapy there was still no apparent lesion over the mental foramen. The maxillary paresthesia had extended to the right lateral orbital and temporal regions. Examinations and roentgenograms one year after therapy revealed no change from the six-month follow-up (Fig. 7).

One month following the one-year examination the patient returned, stating that h had felt "something growing on [his] gums" for the past three weeks. Examination a this time revealed an ulcerated, bluish, fungating, spongy, 1 by 2 cm. mass on the righ retromolar area. Roentgenograms revealed that now the tumor had eroded through the al veolar crest and was invading the soft tissue of the alveolar ridge (Figs. 8, 9, and 10). Th patient was readmitted to the hospital, and the area was biopsied. Again the report indi cated "malignant Schwannoma," the microscopic description being essentially the same a in the previous report. Two days after admission, the patient stated that he had starte

Fig. 7.—Dental film made one year after radiation treatment, showing no essential changes from initial film.

Fig. 8.—Clinical photograph of fungating oral mass which developed thirteen months after initial admission.

having "dizzy spells" and was unable to stand comfortably. The following day he developed typical pains of angina pectoris. A medical consultation was held, and the patient was treated with sublingual nitroglycerin. This apparently relieved his anginal symptoms.

It was decided that the patient, who at this time was confined to a wheel chair, should be given cobalt therapy. After three treatments, however, he developed a resistant staphylococcus infection in his eye and was then isolated in his hospital room. At the present time the patient is comfortable, but his intraoral mass has enlarged.

ADDENDUM

The patient was last examined late in November, 1960. The intraoral tumor mass has ceeded, and exposed bone is causing considerable pain. The neurological findings are still resent, but the patient is doing surprisingly well.

ISCUSSION

In reviewing these cases, it is noted that in the four previous cases and he case presented here the clinical pictures and courses closely parallel one nother. The microscopic pictures in these five cases are also essentially the ame, although a different name was used in some cases. None of these patients

Fig. 9.

Fig. 10.

Fig. 9.—Lateral jaw film made at this time showing erosion through the alveolar crest.
Fig. 10.—Dental roentgenogram of same area.

was noted to have Von Recklinghausen's disease. In the case reported by Lewis and Hart,[41] the patient appeared to have a long history of a "benign" lesion in the lip before overt malignancy was noted. In the case described by Economou and associates[25] there was a history of trauma to the tumor site.

Numerous cases of malignant nerve sheath tumors have been reported in other locations. In order of frequency, these locations are lower extremity, upper extremity, chest wall, abdominal wall, head and neck, buttock, axilla, groin, scalp, and heart. The nerves most frequently involved were the femoral, sciatic, ulnar, median, radial, brachial plexus, anterior cervical, popliteal, lumbosacral plexus, cervical sympathetic, fifth cranial, and scapular region nerves.

The age incidence most commonly ranged from the third to the sixth decade, with no appreciable difference noted between sexes. There were often no symptoms other than the discomfort caused by the mass. When other symptoms are noted, they are usually those of pain, dull ache, and paresthesia along the path of the nerve.

These cases were treated by surgery and/or radiation. This lesion has a marked tendency to recur, and the degree of malignancy increases with each recurrence. The tumor also has a tendency to produce other sites proximally along the course of the nerve. It has been reported that in as many as 20 per cent of these cases pulmonary metastasis will develop.[56] Invasion of the spinal cord,[17] and a case of metastasis to the tail of the pancreas have been reported.[61] Pack and Ariel[54] state that 59.4 per cent of their cases had a five-year survival and that the average life expectancy was seventy-seven months from the time of onset of symptoms.

## SUMMARY

The theories concerning the origin of tumors of nerve sheath have been reviewed, and the multiplicity of terminology of these tumors has been discussed. A review of the literature revealed two cases of malignant nerve sheath tumor of the maxilla and seven involving the mandible and/or the inferior alveolar nerve. A new case of malignant tumor of the inferior alveolar nerve has been presented.

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# ORAL PATHOLOGY

*General Section*

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## PAGET'S DISEASE OF BONE WITH CHARACTERISTIC CHANGES IN THE MANDIBLE AND SELECTIVE SPARING OF THE MAXILLA

*Hilde H. Tillman, D.M.D., West Roxbury, Mass.*

PAGET'S disease of bone, or osteitis deformans, is a curious idiopathic disease of the adult skeleton which is characterized by simultaneous destruction and repair of the involved tissues. It is more common in men than in women, and it appears generally in the fifth and sixth decades. Though it is a localized disorder, the disease can be widespread and involve most of the body. It does not, however, spread from one bone to another across an articular space. It is seen most often in the sacrum, vertebrae, femora, skull, sternum, and pelvis. The facial bones are less frequently involved than those of the skull, and the mandible much less often than the maxilla. In one series, for example, there was involvement of the jaws in 16.6 per cent of the patients; of these, however, only three of twenty-three patients had mandibular disease and only one showed changes in both jaws.[1]

The newly formed bone is coarse and thickened and has a rough surface. This leads to quite extraordinary growth and deformity of the involved parts, which is best and most often seen in the gross enlargement of the skull (Fig. 1). The bone is also of inferior quality and incapable of sustaining normal architectural stresses. This, in turn, results in further distortions, including bowing of the femora and kyphosis of the spine. The involved parts are characteristically warmer than the unaffected adjacent areas as a result of extensive arteriovenous communications in the underlying diseased bone. In the presence of widespread disease, this may lead to strain on the vascular system and eventual heart failure.

Bone pain and spontaneous or pathologic fractures can take place. Still later, characteristic deformities become apparent and occasionally result in an

From the Veterans Administration Hospital, West Roxbury, Massachusetts.

apelike posture and gait. Unless important complications develop, however, Paget's disease can run a long and relatively benign course. The most important and serious complication is the development of osteogenic sarcoma which has its genesis in the abnormal regrowth of bone.[2] Arthritis, atherosclerosis, and hypertensive cardiovascular changes are often encountered. Salivary and renal calculi are relatively common.

The radiologic changes vary according to the stage of the disease, with osteoporosis the first manifestation. Later, as the result of a blending of minute areas of radiolucency and increased density, the typical "cotton wool" appearance becomes apparent. Radiologic evidence is sometimes seen in the jaws before the disorder becomes apparent elsewhere in the skeleton.[3, 4] The

Fig. 1.—Roentgenogram of skull showing gross enlargement and typical "cotton wool" appearance of the osseous tissue.

early changes in the jaw are due to demineralization, with an increase in the number of normal trabeculae giving the appearance of ground glass.[4, 5] When teeth are present, there is a gradual loss of lamina dura in the involved areas of bone.[6] Later the jaws develop a wooly appearance, with the dental roots showing varying degrees of hyperplasia of the cementum. The pulp chambers of these teeth frequently become calcified.

The histologic picture includes abnormalities of the bone and the bone marrow. The osseous tissue changes are characterized by simultaneous destruction and repair, with a mosaic pattern due to the coexistence of various stages of the disorder in the same or neighboring areas. The bone marrow becomes extensively fibrosed.

The blood chemistry determinations, including serum alkaline phosphatase, calcium, and phosphorus, may be normal early in the disease when there has

been little skeletal involvement. More commonly, however, elevation of the alkaline phosphatase is the rule.

ASE REPORT

A 58-year-old retired policeman was admitted to the West Roxbury Veterans Administration Hospital complaining of pain in the right chest and of general bone pain, particularly of the legs and arms. Severe Paget's disease was known to have been present since 1945. The history included gout, arteriosclerotic heart disease with an old myocardial infarct, angina pectoris, and anemia of unknown origin. The patient had experienced multiple rib and finger fractures in the past and had been in congestive heart failure. Increasing dyspnea had been noted during the four months prior to admission.

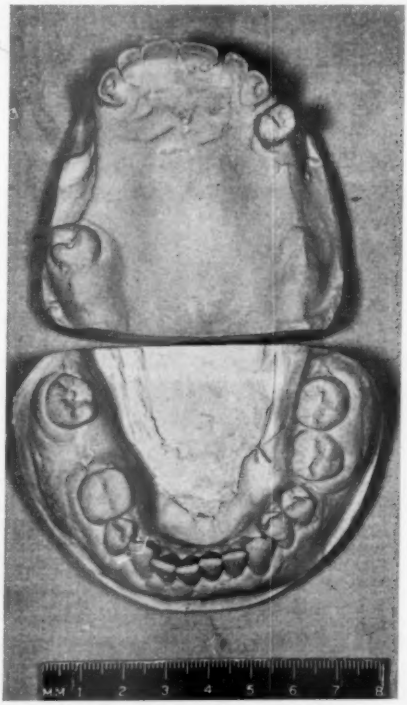


Fig. 2.—Study models of maxilla and mandible showing marked overgrowth of mandible with increased width particularly evident in molar areas.

On physical examination, the patient appeared chronically ill. He was obese and extensively deformed by the skeletal changes of Paget's disease. He had an enlarged skull and showed a marked increase in the anteroposterior diameter of the chest. His arms and legs showed an outward bowing. There was tenderness and crepitus on pressure over the eighth and ninth right ribs at the midaxillary line. The blood pressure was 200/90. Oral examination revealed that the mandible was markedly enlarged, especially in the molar areas.

*Laboratory and X-ray Data.*—The significant laboratory findings included an alkaline phosphatase of 42.5 Bodansky units, a uric acid of 6.9 mg. per cent, and a hematocrit of 35 per cent.

Fig. 3.—Photograph of mouth with teeth in centric relation as registered by patient showing marked enlargement of mandible.

Fig. 4.—Intraoral roentgenogram of mandible showing early demineralization changes in anterior portion of mandible characterized by loss of lamina dura about roots of anterior teeth and trabecular changes. Posterior portion of mandible shows later changes as characterized by cemental hyperplasia of molar roots and calcification of pulp chambers. Surrounding bone appears radiolucent.

A bone survey showed extensive Paget's disease of the entire spine, clavicles, scapulae, humerus, hands, pelvis, femur, skull, and mandible. There was marked calcification of the lower abdominal aorta and pelvic vessels.

The patient was treated conservatively. He was given digitalis, and his right chest was taped with adhesive bandage. The pain was treated with codeine and aspirin, and he was placed on iron-replacement therapy and a low-calorie diet.

A right heart catheterization was performed to assess cardiac output. The results indicated a slightly elevated resting cardiac output which rose moderately with exercise, but the findings did not support the diagnosis of a "high output state."

## DISCUSSION

This case is reported as an interesting example of widespread and intense skeletal involvement by Paget's disease, with selective changes in the facial skeleton. Severe Paget's disease of the mandible is apparently unusual.[1] The jaws, when involved, are said to show typical changes before these changes can be found in other bones.[3, 4] This patient, in retrospect, probably had mandibular disease rather early, since the findings of hypercementosis of the molar roots and calcification of the pulp chambers are evidence of a later stage of Paget's disease.[6] Study models of the maxilla and mandible clearly showed marked overgrowth of the mandible, with increased width particularly evident in the molar areas (Fig. 2). Photographs of the mouth furnish additional evidence of this marked mandibular enlargement (Fig. 3). Intraoral radiographs indicated that different stages of the same disease process can occur side by side (Fig. 4). In the anterior portion of the mandible the early demineralization changes are characterized by the loss of lamina dura about the roots of the lower central incisors and by trabecular changes. A later stage of Paget's disease is seen in the area of the lower molars, where the roots show varying degrees of cemental hyperplasia and calcification of the pulp chambers. The surrounding bone appears radiolucent (Fig. 4).

## SUMMARY

The characteristic bony lesions in Paget's disease are caused by a process of destruction and repair occurring as a localized progressive disorder. In the case reported here, there was widespread skeletal involvement together with characteristic changes in the mandible and selective sparing of the maxilla.

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## ENDODONTICS

*The American Association of Endodontists*

*Captain Warren J. Hedman, Editor*

# INCIDENCE AND DURATION OF PAIN FOLLOWING ENDODONTIC THERAPY

## Relationship to Treatment With Sulfonamides and to Other Factors

*Samuel Seltzer, D.D.S.,* I. B. Bender, D.D.S.,** and Joseph Ehrenreich, M.A., Philadelphia, Pa.*

SULFATHIAZOLE treatment of necrotic pulps, according to Östby,[1] relieves the painful symptoms almost immediately; even the collateral edema is said to disappear in a short time. The use of sulfonamides to reduce the incidence and duration of pain following root canal therapy suggests itself, therefore, as a possibility worth investigating, even though studies of pain are necessarily subjective and difficult to control.

Our purpose was to compare the effects of sulfonamides, antibiotic mixtures, combinations of sulfonamides with antibiotic mixtures, and placebos, some of which are used routinely in root canal therapy, on the incidence and duration of subsequent pain. We also looked for relationships between pain and such factors as age of patient, presence of infection in the tooth, diagnosis, and completeness of instrumentation.

Since placebos can relieve pain by suggestion[2, 3] and the expectations of the dentist can influence both himself and his patients, we used a double-blind technique. Also, we attempted to even out the large number of variables, unavoidable in endodontic practice, by including a large number of patients in the study. We succeeded in recording the reactions of 698 subjects by gaining the active participation of six endodontists*** (at the price, however,

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*Associate Professor, Oral Histology and Oral Pathology, School of Dentistry, University of Pennsylvania.

**Associate Professor, Oral Medicine, School of Dentistry, University of Pennsylvania.

***The endodontists participating in this study were all, with one exception, members of the Philadelphia Endodontic Study Club: Drs. L. Parris, S. Rossman, H. Schilder, W. Soltanoff, D. Snyder, and S. Turkenkoph.

of acquiring a few additional variables). The results obtained from six different observers, although less scientifically "pure," were perhaps more representative of general practice.

## MATERIALS

The materials used were Kynex powder (sulfamethoxypyridazine),* placebos (lactose, made up to resemble Kynex), PSCS (mixture of penicillin, streptomycin, chloromycetin, and sodium caprylate), PBSC (mixture of penicillin, bacitracin, streptomycin, and sodium caprylate), and KSC (mixture of kanamycin, streptomycin, and sodium caprylate).

Kynex was selected as a representative of the newer group of sulfonamide drugs because of its many desirable properties.[4] Also, in a preliminary study of eighty-seven cases, Kynex was compared with sulfathiazole, the drug used by Östby, and the results obtained with the two sulfonamides proved to be virtually identical with respect to incidence and duration of pain. Similarly, a comparison of the results obtained with PBSC and KSC in eighty-two patients showed that there was no significant difference between these mixtures. (Table IV).

## METHOD

Samples of Kynex and of placebos, unlabeled except for a code identification known only to a nonparticipant, were given to six endodontists for trial use in medicating root canals after endodontic procedures. The dentists were instructed to ascertain at the following visit the incidence, severity, and duration of pain experienced by their patients.† Pain was to be graded on a five-point scale, from 0 (no pain) to 4 (intolerable pain). Evaluation of the degree of pain was necessarily a matter of individual judgment, but the kind of medication needed was a rough guide (Table I).

TABLE I. SUMMARY OF INCIDENCE OF VARIOUS DEGREES OF PAIN

| DEGREE OF PAIN | | | | PERCENTAGE | |
|---|---|---|---|---|---|
| DEFINITION IN TERMS OF TREATMENT REQUIRED | | CODE NO. | NO. OF CASES | BASED ON ALL CASES | BASED ON CASES OF PAIN |
| | No pain | 0 | 388 | 59.4 | - |
| | Slight pain (no medication) | 1 | 132 | 20.2 | 49.8 |
| Urgent pain | Moderate (aspirin) | 2 | 71 | 10.9 | 26.8 |
| Urgent pain | Severe (codeine) | 3 | 42 | 6.4 | 15.8 |
| Urgent pain | Interolerable (open and drain; codeine or Demerol; Seconal) | 4 | 20 | 3.1 | 7.5 |
| Totals | | | | | |
| | Slight pain | | 132 | 20.2 | 49.8 |
| | Urgent pain | | 133 | 20.4 | 50.2 |
| | All cases of pain | | 265 | 40.5 | 100.0 |
| | Total number of patients | | 653 | 100.0 | - |

*Kindly supplied by Lederle Laboratories Division, American Cyanamid Company, Pearl River, New York.

†The patients were asked whether they experienced any disturbances following treatment and, if so, how long they lasted. The word "pain" was not used in the interrogation.

The dentists were also asked to record the age of each patient, the diagnosis (acute or chronic pulpitis or pericementitis), the presence of microorganisms in the root canal (plus or minus culture), and the stage of instrumentation (complete or incomplete).

Three of the dentists tested not only the Kynex and the placebo but also the antibiotic mixtures as follows: dentist A, PSCS; dentist B, PBSC or KSC; dentist C, PBSC. The antibiotic mixtures were given in combination with Kynex to some patients and in combination with placebo to others.

The participating dentists were cautioned not to screen out any cases, even the most severe, but to include all patients requiring root canal therapy. Originally, a seventh dentist participated in the study, but his results were discarded when it was learned that he had done some screening.

The data were subjected to statistical analyses. In analyzing the results, it was found expedient to combine some of the categories; otherwise, the data would have been subdivided into numerous cells of classification with too few items in each cell to permit valid statistical inferences. For some purposes, incidence of pain was listed as *none* (grade 0), *slight* (grade 1), or *urgent* (grades 2, 3, and 4). For other purposes, the only division was between *pain* (grades 1 to 4) and *no pain* (grade 0). Similarly, duration of pain was condensed to two categories, *pain for twenty-four hours or less* and *pain for more than twenty-four hours*. In some instances, data obtained by dentists E and F were combined, as they reported relatively few cases.

*Totals.*—Forty per cent of the patients included in the study experienced some degree of pain following root canal therapy. About half experienced slight pain and one-fourth had moderate pain. In the remaining one-fourth, pain ranged from severe to intolerable, mostly the former (Table I).

The duration of pain was usually one day or less, but 15 per cent of the patients (representing 37 per cent of those with pain) reported having pain for more than one day (Table II).

TABLE II. SUMMARY OF DURATION OF PAIN

| DURATION (HOURS) | NO. OF CASES | PERCENTAGE | |
|---|---|---|---|
| | | BASED ON ALL CASES | BASED ON CASES OF PAIN |
| No pain | 388 | 59.4 | - |
| 24 or less | 166 | 25.6 | 62.7 |
| 25-48 | 68 | 10.4 | 25.6 |
| 49-72 | 14 | 2.1 | 5.3 |
| More than 72 | 17 | 2.6 | 6.4 |
| Totals | | | |
| Pain 24 hours or less | 166 | 25.6 | 62.7 |
| Pain more than 24 hours | 99 | 15.1 | 37.3 |
| All cases of pain | 265 | 40.5 | 100.0 |
| Total number of patients | 653 | 100.0 | - |

The results reported by each of the six dentists with respect to incidence of pain were disconcertingly different, varying from 9 per cent to 57 per cent. Pain lasting more than twenty-four hours varied from 4 per cent to 24 per

ent (Table III). It is not possible to say whether the variations are due to differences in dental technique, the kinds of patients constituting each dentist's practice, the dentist's method of interrogating the patients, or his conservatism in evaluating the patients' responses.

In many instances, the patient expects pain following endodontic procedures but does not complain unless interrogated. Severe pain occurs in 11 to 15 per cent of the patients undergoing treatment.

TABLE III. DRUGS APPLIED DURING ENDODONTIC TREATMENT VERSUS SUBSEQUENT PAIN

| DENTIST | DRUGS* | NO. OF PATIENTS TREATED | INCIDENCE OF PAIN† | | | DURATION OF PAIN† | |
|---|---|---|---|---|---|---|---|
| | | | SLIGHT PAIN | URGENT PAIN | ALL CASES OF PAIN | 1-24 HOURS | MORE THAN 24 HOURS |
| A | Placebo | 22 | 2 | 6 (27%) | 8 (36%) | 4 | 4 (18%) |
| | Placebo and antibiotics | 32 | 4 | 13 (41%) | 17 (53%) | 10 | 7 (22%) |
| | Sulfa | 21 | 3 | 10 (48%) | 13 (62%) | 8 | 5 (24%) |
| | Sulfa and antibiotics | 122 | 28 | 47 (39%) | 75 (61%) | 44 | 31 (25%) |
| | Total | 197 | 37 | 76 (39%) | 113 (57%) | 66 | 47 (24%) |
| B | Placebo | 13 | 2 | 2 (15%) | 4 (31%) | 1 | 3 (23%) |
| | Placebo and antibiotics | 91 | 21 | 20 (22%) | 41 (45%) | 24 | 17 (19%) |
| | Sulfa | 18 | 11 | 0 ( 0%) | 11 (61%) | 11 | 0 ( 0%) |
| | Sulfa and antibiotics | 91 | 36 | 11 (12%) | 47 (52%) | 35 | 12 (13%) |
| | Total | 213 | 70 | 33 (15%) | 103 (48%) | 71 | 32 (15%) |
| C | Placebo | 22 | 4 | 1 ( 5%) | 5 (23%) | 4 | 1 ( 5%) |
| | Placebo and antibiotics | 24 | 5 | 3 (13%) | 8 (33%) | 5 | 3 (13%) |
| | Sulfa | 20 | 5 | 2 (10%) | 7 (35%) | 6 | 1 ( 5%) |
| | Sulfa and antibiotics | 24 | 4 | 6 (25%) | 10 (42%) | 4 | 6 (25%) |
| | Total | 90 | 18 | 12 (13%) | 30 (33%) | 19 | 11 (12%) |
| D | Placebo | 56 | 3 | 11 (20%) | 14 (25%) | 5 | 9 (16%) |
| | Sulfa | 50 | 4 | 3 ( 6%) | 7 (14%) | 4 | 3 ( 6%) |
| | Total | 106 | 7 | 14 (13%) | 21 (20%) | 9 | 12 (11%) |
| E | Placebo | 12 | 1 | 2 (17%) | 3 (25%) | 3 | 0 ( 0%) |
| | Sulfa | 13 | 2 | 1 ( 8%) | 3 (23%) | 2 | 1 ( 8%) |
| | Total | 25 | 3 | 3 (12%) | 6 (24%) | 5 | 1 ( 4%) |
| F | Placebo | 31 | 1 | 1 ( 3%) | 2 ( 6%) | 1 | 1 ( 3%) |
| | Sulfa | 36 | 2 | 2 ( 6%) | 4 (11%) | 1 | 3 ( 8%) |
| | Total | 67 | 3 | 3 ( 5%) | 6 ( 9%) | 2 | 4 ( 6%) |
| Totals for 3 dentists (A, B, C) | Placebo | 57 | 8 | 9 (16%) | 17 (30%) | 9 | 8 (14%) |
| | Placebo and antibiotics | 147 | 30 | 36 (24%) | 66 (45%) | 39 | 27 (18%) |
| | Sulfa | 59 | 19 | 12 (20%) | 31 (52%) | 25 | 6 (10%) |
| | Sulfa and antibiotics | 237 | 68 | 64 (27%) | 132 (56%) | 83 | 49 (21%) |
| | Total | 500 | 125 | 121 (24%) | 246 (49%) | 156 | 90 (18%) |
| Totals for all 6 dentists (A, B, C, D, E, F) | Placebo | 156 | 13 | 23 (15%) | 36 (23%) | 18 | 18 (12%) |
| | Placebo and antibiotics | 147 | 30 | 36 (24%) | 66 (45%) | 39 | 27 (18%) |
| | Sulfa | 158 | 27 | 18 (11%) | 45 (28%) | 32 | 13 ( 8%) |
| | Sulfa and antibiotics | 237 | 68 | 64 (27%) | 132 (56%) | 83 | 49 (21%) |
| | Grand total | 698 | 138 | 141 (20%) | 279 (40%) | 172 | 107 (15%) |

*Sulfa = sulfonamides (Kynex); see text for particular ingredients of antibiotic mixtures.
†Number of cases. Percentages (rounded off to whole numbers) are based on total number of patients treated with the particular drug.

*Drugs.*—It would be misleading to add up the number of cases in which pain occurred after the use of each drug without allowing for the differences in the results reported by each dentist. Thus, dentist A, reporting the highest incidence of pain, contributed a disproportionate number of cases treated with sulfonamide-antibiotic combinations. The effect was to make it seem that treatment with such combinations was followed by the greatest incidence of pain (56 per cent) and treatment with placebos the lowest incidence of pain (23 per cent) (Table III).

Instead of combining the results of all the dentists by simple addition, we calculated the probabilities for the results of each dentist separately. Then we combined the results according to a method developed by Fisher, who showed that for different types of experiments bearing on the same treatment, $-2 \log P$ is distributed as chi-square with 2 degrees of freedom.[5]

Table III shows the incidence and duration of pain following use of each drug, as reported by each dentist, in terms of number of cases and percentage. The probabilities that differences in results obtained with each pair of drugs

TABLE IV. SUMMARY OF STATISTICAL SIGNIFICANCE OF DATA ON INCIDENCE, SEVERITY, AND DURATION OF PAIN AS AFFECTED BY VARIABLES OF CLINICAL CONDITION AND TREATMENT

| VARIABLES COMPARED | ATTRIBUTES CONSIDERED | $\chi^2$ | D.F. | P |
|---|---|---|---|---|
| Placebo, sulfa | Incidence | 12.37* | 10 | 0.26 |
| Placebo, sulfa | Duration | 2.92 | 1 | 0.09 |
| Placebo, antibiotics | Incidence | 4.98* | 6 | 0.55 |
| Placebo, antibiotics | Urgent versus all other | 2.2 | 4 | 0.63 |
| Placebo, antibiotics | Duration | 2.10 | 1 | 0.16 |
| Sulfa, sulfa and antibiotics | Incidence | 1.37* | 6 | 0.97 |
| Sulfa, sulfa and antibiotics | Duration | 0.67 | 1 | 0.45 |
| Sulfa, antibiotics | Duration | 1.29 | 1 | 0.31 |
| No antibl., PSCS, KSC | Incidence and severity | 6.01 | 4 | 0.211 |
| Sulfathiazole versus Kynex | Incidence and severity | | | N. S. |
| Sulfathiazole versus Kynex | Duration | | | N. S. |
| Age: $<$ 21 versus $>$ 21 years | Incidence and severity | 21.72 | 2 | $<$ 0.005 Signif. |
| Age: $<$ 21 versus $>$ 21 years | Duration | 0.41 | 1 | 0.53 |
| Positive versus negative culture (before treatment) | Incidence and severity | 4.76 | 2 | 0.095 |
| Positive versus negative culture (before treatment) | Duration | 0.001 | 1 | $>$ 0.950 |
| Acute versus chronic | Incidence and severity | 14.46 | 2 | $<$ 0.005 Signif. |
| Acute versus chronic | Duration | 3.40 | 1 | 0.07 |
| Complete versus incomplete instrumentation | | | | N. S. |
| Complete versus incomplete instrumentation | | | | N. S. |

*Symbols and terms:*

$\chi^2$ = Chi square.

D.F. = Degrees of freedom.

P = Probability of a greater difference occurring by chance.

N.S. = Obviously not significant for inspection of percentages.

Signif. = Statistically significant.

Incidence = Total number of cases with pain versus cases without pain. Duration = pain $<$ 24 hours versus pain $>$ 24 hours. Incidence and severity = No pain, slight pain, and urgent pain in a three-way comparison.

*Calculated from the results of each dentist taken separately and combined according to the principle that combined chi square $= -2 \log_e P$ with D.F. $= 2 \times$ number of separate sets of data. Values of P for the individual dentists were, for placebo versus sulfa, (A) 0.075, (B) 0.20, (C) 0.57, (D) 0.24, (E-F) 0.99+; placebo versus antibiotics, (A) 0.25, (B) 0.50, (C) 0.66; placebo versus antibiotics (urgent pain versus all other cases), (A) 0.57, (B+C) 0.58; sulfa versus sulfa and antibiotics, (A) 0.85, (B) 0.66, (C) 0.90. None of these P values, individually, were significant.

re due to chance are listed in Table IV. A breakdown of the probabilities as determined from the results reported by each dentist separately is given in the footnotes to Table IV.

*Sulfonamides Versus Placebos.*—In over-all results, the incidence of pain after the use of sulfonamides was 28 per cent, and after the use of placebos it was 23 per cent. If only urgent pain were considered, the results would be 11 per cent and 15 per cent, respectively. The probabilities, obtained by combining the results reported by each dentist taken separately according to the method described in the previous paragraph, indicate that there is no statistically significant difference between sulfonamides and placebos with respect to incidence of pain.

Cases in which pain lasted for more than a day were as follows: for sulfonamides, 8 per cent of all cases; for placebos, 12 per cent of all cases. If percentages were based not on all cases but only on cases in which pain was experienced, the results would be, for sulfonamides, thirteen out of forty-five cases (29 per cent) and, for placebos, eighteen out of thirty-six cases (50 per cent). These last results are not statistically significant at the conventional 95 per cent level, but nevertheless there is less than one chance in ten that the results are accidental.

The results indicate, then, that sulfonamides do not materially reduce the incidence or severity of pain but may possibly shorten the duration of pain.

*Antibiotics Versus Placebo.*—The incidence of pain is actually greater with antibiotics than with placebo, according to the data of each dentist separately or combined. The combined results for the three dentists who treated root canals with antibiotics were as follows: antibiotics, 45 per cent incidence of pain; placebos, 30 per cent incidence. Nevertheless, the results fell short of statistical significance, whether they are calculated directly ($P = 0.09$) or by combining separate probabilities ($P = 0.55$).

The incidence of urgent pain also was higher when antibiotics were used: 24 per cent as compared to 16 per cent for the three dentists. Again, the results were not statistically significant.

The duration of pain for more than a day, expressed as a percentage of the total number of cases in which pain was experienced by the patients of the three dentists, was about the same following antibiotics (41 per cent) as following placebos (47 per cent).

The possibility is suggested, although not proved, that one or more constituents of the antibiotic mixtures are irritating and cause pain. Studies by Schilder and Amsterdam[6] also point to the irritating quality of some of these antibiotic mixtures.

*Sulfonamides Versus Sulfonamides Plus Antibiotics.*—The combination of sulfonamides with antibiotics is followed by an incidence of pain (56 per cent) and of urgent pain (27 per cent) not significantly different from that following the use of sulfonamides alone (52 per cent and 20 per cent, if the comparison is restricted to the same three dentists who worked with antibiotics). The duration of pain, likewise, is not significantly different.

*Sulfonamides Versus Antibiotics.*—A comparison of the effects of sulfonamides and antibiotics, confined to the data supplied by the three dentists who worked with both, shows an incidence of pain in 52 per cent of the patients given sulfonamides and in 45 per cent of those given antibiotics. Urgent pain was experienced by 20 per cent of the patients given sulfonamides and 24 per cent of those given antibiotics. Pain lasted more than one day in 20 per cent of the sulfonamide-treated patients who experienced pain and in 41 per cent of the antibiotic-treated patients who had pain. Sulfonamides seem to have an advantage with respect to degree of pain and duration of pain, but calculations fail to show statistical significance.

*Age of Patients.*—Table V (on age of patients in relation to pain) and the remaining tables show the combined results reported by all the dentists, as the pertinent variables seem to be randomly distributed.

TABLE V. AGE OF PATIENT VERSUS PAIN AFTER ENDODONTIC TREATMENT

| AGE (YEARS) | NO. OF PATIENTS | INCIDENCE OF PAIN* | | | DURATION OF PAIN* | |
|---|---|---|---|---|---|---|
| | | SLIGHT PAIN | URGENT PAIN | ALL CASES OF PAIN | < 24 HOURS | > 24 HOURS |
| < 11 | 8 | 2 | 0 ( 0%) | 2 (25%) | 2 | 0 ( 0%) |
| 11-15 | 52 | 3 | 6 (12%) | 9 (17%) | 7 | 2 ( 4%) |
| 16-20 | 53 | 6 | 7 (13%) | 13 (25%) | 8 | 5 ( 9%) |
| 21-30 | 90 | 16 | 22 (24%) | 38 (42%) | 23 | 15 (17%) |
| 31-40 | 150 | 37 | 31 (21%) | 68 (45%) | 40 | 28 (19%) |
| 41-50 | 153 | 35 | 38 (25%) | 73 (48%) | 46 | 27 (18%) |
| 51-60 | 116 | 27 | 21 (18%) | 48 (41%) | 33 | 15 (13%) |
| 61-70 | 25 | 6 | 7 (28%) | 13 (52%) | 7 | 6 (24%) |
| > 70 | 6 | 0 | 1 (17%) | 1 (17%) | 0 | 1 (17%) |
| Totals | | | | | | |
| < 21 | 113 | 11 | 13 (12%) | 24 (21%) | 17 | 7 ( 6%) |
| 21–70+ | 540 | 121 | 120 (22%) | 241 (45%) | 149 | 92 (17%) |
| All ages | 653 | 132 | 133 (20%) | 265 (41%) | 166 | 99 (15%) |

*Number of cases. Percentages (to nearest whole number) are based on total number of patients in the age category.

Inspection of the data on incidence of pain shows a sharp increase beginning at about age 21 (pain occurred in 21 per cent of the patients under 21 and in 45 per cent of those over 21). Calculations based on the number with no pain, slight pain, and urgent pain show that the difference in the incidence and severity of pain reported by these two groups is highly significant ($P = < 0.005$). On the other hand, there is no significant difference in the duration of pain (Tables IV and V). A similar relationship between age and incidence of pain has been found by Herzberg.[7]

*Chronic or Acute Pulpitis and Pericementitis.*—The condition of the tooth was diagnosed as acute pulpitis or acute pericementitis in almost half the cases. The incidence of pain was about the same for pulpitis as for pericementitis (48 per cent and 54 per cent, respectively). On the other hand, there was a distinct difference in incidence of subsequent pain (following medication) as between the acute condition (49 per cent) and the chronic (34 per cent. This difference was statistically significant ($P = < 0.005$). When pain did occur, its duration was almost, but not quite, significantly different for the acute and chronic conditions (Tables IV and VI).

TABLE VI. DIAGNOSIS VERSUS PAIN

| DIAGNOSIS | NO. OF PATIENTS | INCIDENCE OF PAIN* | | | DURATION OF PAIN* | |
|---|---|---|---|---|---|---|
| | | SLIGHT PAIN | URGENT PAIN | ALL CASES OF PAIN | < 24 HOURS | > 24 HOURS |
| Acute pulpitis | 196 | 50 (26%) | 43 (22%) | 93 (48%) | 50 (26%) | 43 (22%) |
| Acute pericementitis | 99 | 27 (27%) | 25 (26%) | 52 (53%) | 39 (39%) | 13 (13%) |
| Chronic pulpitis or pericementitis | 326 | 60 (18%) | 51 (16%) | 111 (34%) | 68 (21%) | 43 (13%) |
| Other | 36 | 6 (17%) | 6 (17%) | 12 (33%) | 9 (25%) | 3 ( 8%) |
| Totals | 657 | 143 (22%) | 125 (19%) | 268 (41%) | 166 (25%) | 102 (16%) |

*Number of cases. Percentages (to nearest whole number) are based on the total number of cases in the diagnostic category.

*Presence of Infection.*—Positive cultures were obtained from about two-thirds of the teeth before the canal was entered. However, the incidence and duration of pain were only a little greater when the cultures were positive; the difference was not great enough to be statistically significant (Tables IV and VII).

TABLE VII. INFECTION BEFORE CANAL IS ENTERED VERSUS SUBSEQUENT PAIN

| CULTURE | NO. OF PATIENTS | INCIDENCE OF PAIN* | | | DURATION OF PAIN* | |
|---|---|---|---|---|---|---|
| | | SLIGHT PAIN | URGENT PAIN | ALL CASES OF PAIN | < 24 HOURS | > 24 HOURS |
| + | 422 | 97 | 88 (21%) | 185 (44%) | 116 | 69 (16%) |
| − | 205 | 41 | 31 (15%) | 72 (35%) | 46 | 26 (13%) |
| Totals | 627 | 138 | 119 (19%) | 257 (41%) | 162 | 95 (15%) |

*Number of cases. Percentages (to nearest whole number) are based on the total number of cases having the given type of culture.

*Instrumentation.*—Some patients were reported upon at a stage when the instrumentation was incomplete and others when the instrumentation was complete, but the incidence of pain was virtually the same (41 per cent and 39 per cent, respectively) in both classes (Table VIII).

TABLE VIII. INSTRUMENTATION VERSUS INCIDENCE OF SUBSEQUENT PAIN

| INSTRUMENTATION | NO. OF PATIENTS | INCIDENCE OF PAIN* | | |
|---|---|---|---|---|
| | | SLIGHT PAIN | URGENT PAIN | ALL CASES OF PAIN |
| Complete | 407 | 90 | 77 (19%) | 167 (41%) |
| Incomplete | 239 | 44 | 48 (20%) | 92 (38%) |
| Totals | 646 | 134 | 125 (19%) | 259 (40%) |

*Number of cases. Percentages (to nearest whole number) are based on total number of patients in the instrumentation category.

## SUMMARY AND CONCLUSIONS

A double-blind study was conducted by six endodontists on 698 patients to determine the effects of sulfonamide treatment and other factors on the incidence, severity, and duration of pain following root canal therapy. Drug pairs compared were sulfonamides versus placebo, antibiotic mixtures versus placebo, sulfonamides versus antibiotic mixtures, and sulfonamides versus a combination of sulfonamides with antibiotics.

1. Forty per cent of all patients experienced pain; in half of these patients the discomfort was slight.

2. The incidence of pain reported for the patients of each dentist varied greatly (from 9 per cent to 57 per cent).

3. There was no statistical difference in the incidence or severity of pain, regardless of which pair of drugs was compared.

4. Differences in the duration of pain possibly exist for only one pair of drugs—sulfonamides versus placebo—and even here the differences are not quite significant statistically. Of the patients treated with sulfonamides, 8 per cent had pain lasting more than one day (29 per cent of the cases of pain), whereas pain of that duration was felt by 12 per cent of the patients (50 per cent of the cases of pain) who had been treated with placebos.

5. Patients younger than 21 years had a significantly lower incidence of pain (21 per cent) than those who were 21 or older (45 per cent). There were no important differences in the duration of the pain for the two age groups.

6. Incidence, but not duration, of pain was significantly greater following treatment in patients with acute pulpitis or pericementitis than in those with chronic pulpitis or pericementitis.

7. Positive cultures were obtained from about two-thirds of the teeth before the canal was entered. The incidence and duration of pain were only a little greater when the cultures were positive. The difference was not enough to reach statistical significance.

8. The stage of instrumentation (complete or incomplete) was not related to either incidence or duration of pain.

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# A STANDARDIZED ENDODONTIC TECHNIQUE UTILIZING NEWLY DESIGNED INSTRUMENTS AND FILLING MATERIALS

*John I. Ingle, D.D.S., M.S.D.,* Seattle, Wash.*

## INTRODUCTION

THE acceptance of root canal therapy by the profession and the public has been a very slow process. The laity has long held doubts about the advisability of retaining the pulpless tooth—doubts based in part upon fears of focal infection. The profession, being generally more sophisticated, is no longer blindly obeisant to the theory of focal infection. Certain members of the profession, however, still retain some doubts concerning the ultimate success to be obtained by endodontic treatment. These professional doubts are not founded on facts, for today it may be reported that well-executed root canal therapy is as successful as any other kind of dental treatment.

Success in endodontics, as in the other disciplines of dentistry, demands careful attention to case selection. This must be followed by meticulous management of the endodontic triad of canal preparation, sterilization, and obturation.[1] Not all endodontically treated cases are successful; nor, for that matter, should we expect them to be. However, the endodontist does expect about 95 per cent success, and when the occasional failure occurs he is dismayed by the result.

Success in any endeavor is dependent upon a well-planned approach coupled with a careful execution of procedure. In spite of prior planning, unforeseen variables which may eventually contribute to failure are sometimes introduced. One learns not so much by gloating over successes as by judiciously analyzing his failures. In so doing, one may determine wherein he made mistakes in judgment or mistakes of execution which ultimately led to failure. Logically, then, correction of an error in judgment and/or technique should increase success.

## ANALYSIS OF SUCCESS AND FAILURE

To determine the rate of success and, conversely, the rate and cause of failure, the Department of Endodontics of the University of Washington, a

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*Professor and Executive Officer, Department of Periodontics and Endodontics, University of Washington School of Dentistry.

decade ago, began an analytical study of all root canal therapy cases treated in the endodontic clinic and, in addition, of cases treated by members of the teaching staff. Out of this study has come some very interesting information which will be reported in a future paper. For the present, however, it may be stated that endodontic failure occurs 5.55 per cent of the time. Analysis of the endodontic failures has led to refinements in case selection and technique which have, in turn, ensured greater success.

Fig. 1.—Incomplete obturation of the root canal accounts for 58.65 per cent of all endodontic failures. Root resection in this case could in no way overcome the toxic irritants arising from the partially filled canals.

## ENDODONTIC FAILURE

Documentation of the causes of endodontic failure was the most significant development of the Washington study. Case analysis revealed improper obturation of the root canal space as the leading cause of failure. It was found that 58.65 per cent of 104 endodontic failures were related to the poorly filled root canal (Fig. 1). The next greatest reason for failure was related to root perforation. However, these operative errors accounted for only 9.62 per cent of the unsuccessful cases (Fig. 2).

Thus, it would appear that poor root canal obturation is by far the greatest cause of endodontic failure, with incorrect canal instrumentation the next most important aspect. Actually, though, the problem is broader than this statement would indicate. Total obturation is dependent, in turn, upon careful coronal and radicular cavity preparation as well as proper case selection and the correct use of filling materials.

The profession is eternally grateful to the late Dr. Francisco M. Pucci, of Montevideo, Uruguay, for his monumental contributions in the field of endodontics.[2] It was Pucci who first pointed out the necessity for meticulous intracoronal preparation as a basis for ultimate success in intraradicular preparation and obturation. Pucci's lifetime of devotion to his profession has not been fully appreciated in North America or, for that matter, in Central Europe. Many of us realize, however, that no matter what aspect of the subject is being discussed, Pucci usually said it first and better.

Fig. 2.—Improper canal instrumentation leading to perforation accounts for 9.62 per cent of the endodontic failures. This may be related to a limited-access cavity or total lack of uniformity in the enlarging instruments.

Leaning heavily on Pucci, I previously have discussed some of our problems, including the importance of correct endodontic cavity preparation[3] and canal obturation.[1-4]

## INSTRUMENT UNIFORMITY

Recent improvements in operative endodontics and canal sterilization leave as the major endodontic problem today the total lack of uniformity and standardization of endodontic instruments and filling materials.[5] The current shortcoming of instrument and filling material uniformity may be expressed in a number of ways:

1. There is at present no uniform numbering system for both instruments and materials. The arbitrary nomenclature of "fine," "medium," and "coarse" is sometimes used, and the numerical (1-2-3-4-5-6) system also applies.
2. There is no uniform system for determination of instrument size, the presently available sizes being entirely arbitrary.
3. There is little uniformity in manufacturing control, with the result that instruments of the same number will frequently vary greatly in perimeter.

4. There is no uniformity to the progression from one instrument size to the next; nor is there any formula for determining the increment of progression.
5. There is no uniform correlation of instruments and filling materials in terms of size and shape.
6. There has been no serious attempt to replace the time-honored plastic gutta-percha with a modern plastic that is better adapted to the problem and more easily controlled in manufacture.

In short, there has been an almost total absence of research and development in this area of endodontics.

### OPERATIVE ENDODONTICS

Ideally, one should be able to prepare a cavity within the root canal and thereafter place a filling in the cavity with no intermediate trial-and-error procedures. Seidler[6] has described the ideal intraradicular cavity as being round and tapered, with a minimum opening at the foramen. This style of cavity may be prepared rapidly and efficiently by means of reamers to enlarge the apical

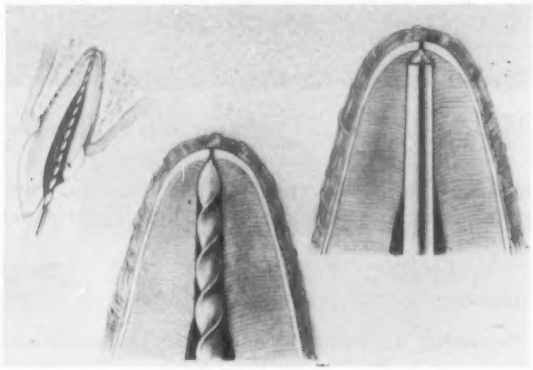

Fig. 3.—*Left:* Preparation with reamers of the "round, tapered apical cavity with a minimal opening at the foramen." The cavity is terminated 0.5 mm. from the apex. The ovoid portion of the canal is finished with files. *Right:* The apical cavity is obturated completely by a plastic or silver point cemented to place. Cement fills the apical triangle cut by the point of the reamer. The remainder of the canal is obturated by spreading the initial point aside and adding multiple fine gutta-percha points. (Courtesy Dr. Lester E. Breese, University of California.)

3 or 4 mm. of the canal (Fig. 3) and files to finish the ovoid portion of the canal.[1] In the small canals of molars and premolars, reamers alone will usually complete the entire operation (Fig. 4). Operating in the presence of the chelating agent, ethylenediaminetetraacetic acid (EDTA), greatly simplifies the procedure. As long as the root canal is comparatively large and large instruments may be used, this method of canal preparation is very efficient and very rapid.

Trouble with this technique develops when the root canal lumen is narrow and curved and the enlarging instruments fit the canal tightly. Under these

circumstances, when a variance in instrument size is encountered the instruments either bind in the canal and are broken or shelf formation or perforation of the root develops.

The second siege of trouble is encountered when one attempts to fit a silver or gutta-percha point to the round-tapered cavity which has been prepared.[5] The presently available filling materials are usually of a larger size than the enlarging instruments of the same number. Consequently, by trial and error, the dentist chooses a filling point of smaller size which, in turn, may not adequately obturate the canal space when cemented to place. Moreover, the shape of the preformed filling materials differs greatly from the shape of the instruments. Because the filling materials are so inefficient, it is not unusual for the final result to be somewhat less than adequate.

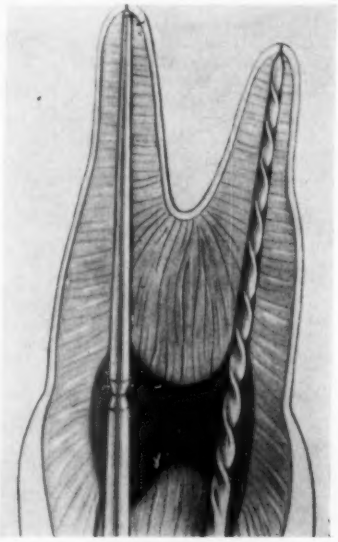

Fig. 4.—A silver point, newly standardized to the size and taper of the reamer last used in preparation, will, when cemented to place, assure total obliteration of the root canal space. Notice how the silver point has been weakened by notching with a carborundum disk prior to seating. The cut is made at a mark 1 mm. above the orifice so that, if necessary, the point may be later removed. The excess is broken away by "worrying" the butt end back and forth until it breaks at the notch. Instrumentation of the small canals in molars and premolars may be accomplished entirely with reamers, ensuring a round, tapered preparation.

The solution to the problem lies in the development of completely uniform endodontic instruments and filling materials. Uniformity can be promoted only by an acceptable formulation for all involved supplies.

## STANDARDIZED INSTRUMENTS

Fortunately, one of the dental manufacturers became interested in the uniformity problem and a now complete system of formulated endodontic instruments and filling materials has been developed and tested for uniformity by microcomparator.[7]

The following standardization has been accomplished:

1. A formula for the diameter and taper in each size of instrument and filling materials has been agreed upon.
2. A formula for a graduated increment in size from one instrument to the next has been developed.
3. A new instrument-numbering system based upon instrument diameter is now being used.
4. Gutta-percha, as a filling material, is being re-evaluated with a strong possibility that it will be replaced by a newer plastic.

As demonstrated by Table I, a new numbering system using numbers from 10 to 100 has been developed. The number of the instrument is not just an arbitrary figure but is based upon the diameter of the instruments in tenths

TABLE I. NUMBERING SYSTEM AND DIAMETER

| SPECIFICATIONS | | FILES AND REAMERS | | SILVER AND PLASTIC POINTS | |
|---|---|---|---|---|---|
| | | DIAMETER* | | DIAMETER† | |
| NEAREST OLD NUMBER | NEW NUMBERS | $D_1$ (MM.) | $D_2$ (MM.) | $D_1$ (MM.) | $D_2$ (MM.) |
| 0 | 10 | 0.1 | 0.4 | | |
| 1 | 15 | 0.15 | 0.45 | | |
| 2 | 20 | 0.2 | 0.5 | | |
| 3 | 25 | 0.25 | 0.55 | 0.25 | 0.55 |
| 4 | 30 | 0.3 | 0.6 | 0.3 | 0.6 |
| — | 35 | 0.35 | 0.65 | 0.35 | 0.65 |
| 5 | 40 | 0.4 | 0.7 | 0.4 | 0.7 |
| — | 45 | 0.45 | 0.75 | 0.45 | 0.75 |
| 6 | 50 | 0.5 | 0.8 | 0.5 | 0.8 |
| — | 55 | 0.55 | 0.85 | 0.55 | 0.85 |
| 7 | 60 | 0.6 | 0.9 | 0.6 | 0.9 |
| 8 | 70 | 0.7 | 1.0 | 0.7 | 1.0 |
| 9 | 80 | 0.8 | 1.1 | 0.8 | 1.1 |
| 10 | 90 | 0.9 | 1.2 | 0.9 | 1.2 |
| 11 | 100 | 1.0 | 1.3 | 1.0 | 1.3 |

Specifications for newly designed and standardized root canal instruments and filling materials. Note the new numbering system based upon the tip diameter of the instruments in tenths of millimeters. The progressive increment in size from one instrument to the next is 0.05 mm. through No. 60, after which the instruments progressively gain 0.1 mm. through No. 100. No. 120 and No. 140 instruments are also now available.

The standardized taper of all instruments is achieved by an increase of 0.3 mm. from $D_1$ to $D_2$, a point 16 mm. up the shaft. Note the minus tolerances for the points. This slight difference allows for the cementing medium and compressibility of dentine during enlarging.

*Tolerances $D_1$ + 0.00 mm. −0.00 mm. $D_2$ + 0.00 mm. −0.00 mm.

†Tolerances $D_1$ + 0.00 mm. −0.01 mm. $D_2$ + 0.00 mm. −0.01 mm.

(From Ingle, J. I.: In Grossman, L. I. [editor]: Transactions of the Second International Conference on Endodontics, Philadelphia, 1958, University of Pennsylvania.)

of millimeters at the beginning of the tip of the blades, a point called $D_1$ (Fig. 5). A No. 10 instrument, for example, is 0.1 mm. at $D_1$, whereas a No. 15 is 0.15 mm., a No. 25 is 0.25 mm., and a No. 100 is 1.0 mm.

The progressive increment in size from one instrument to the next is 0.05 mm. As Table I shows, there is an increase of 0.05 mm. in size from the No. 10 instrument through the No. 60 instrument. Thereafter, the increase in size is 0.1 mm. from the No. 60 instrument through the No. 100 instrument. There are now fifteen instrument sizes in both files and reamers.

The cutting blades of each instrument extend up the shaft 16 mm. from point $D_1$, and this larger diameter is called $D_2$. The diameter of each instrument at $D_2$ is 0.3 mm. greater than the diameter at $D_1$ (Table I). Because of the standardized length of blade and the standardized diameter increase from $D_1$ to $D_2$, a formula may be established for uniform taper:

$$\frac{D_2 - D_1}{\text{Length between } D_2 \text{ and } D_1} = \frac{0.3 \text{ mm.}}{16.0 \text{ mm.}} = 0.01875 \text{ mm. per millimeter}$$

or an increase in each instrument of 18.75 microns per mm. of length. In short, each instrument, no matter what size, will have the same taper of shaft.

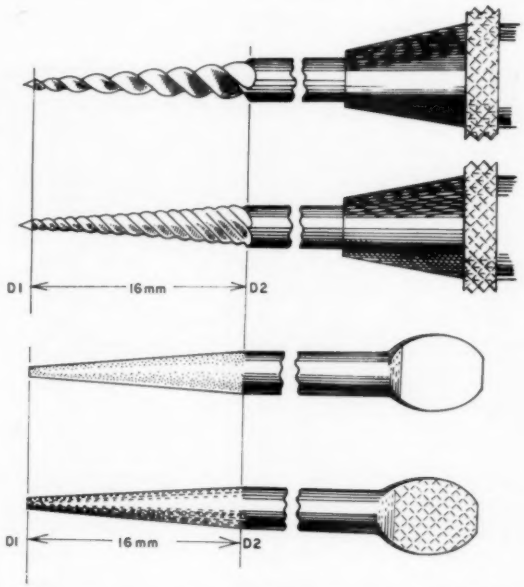


Fig. 5.—Newly designed endodontic instruments and filling materials, uniform throughout in size, taper, and length. The diameter of the instrument in tenths of millimeters at $D_1$ determines the number of the instrument (10 to 100). Point $D_2$ is standardized at 16 mm. from $D_1$. The increase in size from $D_1$ to $D_2$ is 0.3 mm. in every size. The taper of every instrument is, therefore, standardized.

(From Grossman, L. I. [editor]: Transactions of the Second International Conference on Endodontics, Philadelphia, 1958, University of Pennsylvania.)

The standardization of these instruments and filling materials is an accomplished fact. The supplies are now available from dental supply depots throughout the world. The silver points are identical to the cutting instruments in number, taper, and increment. On the other hand, the over-all diameter of the silver points has been made about 9 microns (0.009 mm.) smaller than that of the enlarging instruments. This difference in size compensates for the thickness of the cementing medium, dentine compressibility, and the common failure on the part of the operator to utilize the instrument to the utmost.

## DISCUSSION

What advantages will accrue to the profession and to the endodontic patient from these newly standardized materials?

First of all, one may surmise that endodontic cavity preparation will improve materially. There should be fewer broken instruments and root perforations, which account for about 10 per cent of our endodontic failures. Furthermore, a vast improvement in obturation will develop, for *accurately* adapted filling materials may now be cemented to place in *accurately* prepared canal cavities (Figs. 3 and 4). Obviously, the new materials will not solve all the endodontic problems. In the hands of the careful operator, however, these new materials should ensure a better grade of service for the patient, with an assurance of greater success and the bonus of a saving in operating time (Fig. 6).

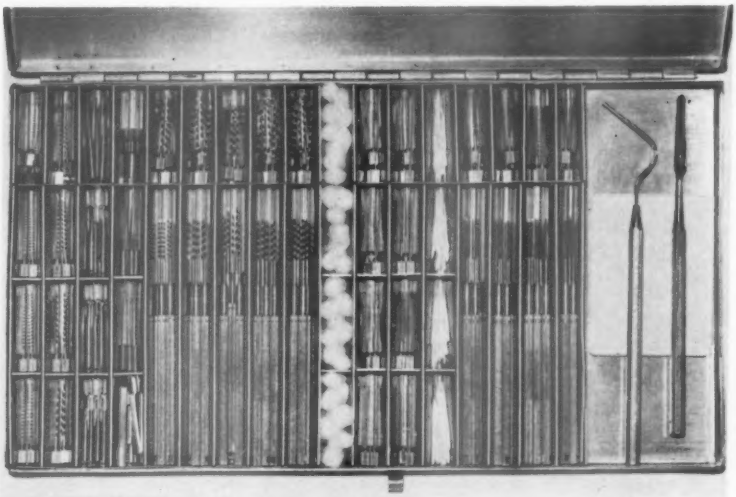

Fig. 6.—A standardized endodontic technique requires complete organization in operation. The stainless instrument case contributes greatly to this organization. Here each instrument has its own space.

The case has a hinged cover and may be sterilized in a dry-heat oven. Instruments and supplies remain sterile therein and are removed with operating pliers only as needed.

The newly standardized endodontic instruments and filling materials will prove to be a major advance in the profession of dentistry.

## CONCLUSIONS

1. The major causes of endodontic failure are incomplete obturation of the canal space and incorrect canal instrumentation.

2. Many endodontic failures are related to the total lack of uniformity or standardization in endodontic instruments and filling materials.

3. Endodontic instruments and filling materials which have been standardized in diameter, taper, and graduated size increment are now available.

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## RESEARCH

# ORAL ASPECTS OF MONGOLISM

## Part I. Periodontal Disease in Mongolism

*M. Michael Cohen, D.M.D., Richard A. Winer, A.B., D.M.D., Stanley Schwartz, D.M.D., and Gerald Shklar, D.M.D., M.S., Boston, Mass.*

### INTRODUCTION

RECENT advances in cytogenetics by investigators in Great Britain, France, and the United States have suggested a chromosomal aberration in mongolism.[1-8] For almost a century it was thought that the constellation of anomalies observed in this condition originated during the sixth to the ninth week of gestation.[9, 10]

Mongolism is always associated with some degree of mental subnormality. Approximately eighty-six anomalies and functional disorders of this syndrome have been described.[11] Two of the most easily recognizable characteristics are cranial and facial deformities (Fig. 1). Other anomalies observed in persons with mongolism are large anterior fontanel and open sutures, small slanting orbits, epicanthal folds, cataracts, strabismus, short nose and small mouth, constricted facial features, short neck, fetal ear patterns, compressed brain, small frontal lobes, underdeveloped cerebellum, four-finger line, gap between the great and second toes, in-curved little fingers caused by maldevelopment of second phalanx, high arched palate, macroglossia and fissured tongue, prognathism, sexual underdevelopment (small penis, undescended testes, and abnormal scrotum in the male; protruding clitorus, underdeveloped labium minus, and large pouchy labium major in the female), patent foramen ovale and septal defects, muscular hypotonia, and hypermobility of the joints.[12-50]

The oral conditions previously described have been limited principally to the dentition, palate, and tongue. Two brief reports by Dow[51] and Nash[52] emphasized the prevalence of severe periodontal disease in young mongoloid children. In the present investigation a group of 100 young mongoloid patients

From Tufts University School of Dental Medicine.

This work was supported by Grant D760 of the National Institutes of Health, United States Public Health Service.

(fifty seven males and forty three females, aged 1 to 30 years)* were observed for the prevalence of periodontal disease as well as other anomalies in the oral cavity.

PROCEDURE

*Clinical Examination.*—Intraoral color transparencies of each patient were taken. With a mouth mirror, a sharp explorer, and good lighting, one examiner (R. A. W.) recorded all decayed, missing, and filled deciduous and permanent teeth. The size and eruption of teeth, particularly the lateral incisors, the occlusal relationship of the jaws, and the morphology of the palate and tongue were carefully observed by a second examiner (M. M. C.) (Fig. 2). The periodontal examination was made by both examiners, and gingival disease observed clinically was rechecked with the color transparencies taken at the beginning of each examination.

Fig. 1.—Facial features of a mongoloid child.

*Roentgenographic Examination.*—Many of the patients with low I. Q.'s were unmanageable, and premedication was prescribed.† In some of the difficult cases it was possible to take the roentgenograms with the patient in a standing position. Roentgenograms were taken of each patient with ultraspeed film and an x-ray machine operating at 90 KVP, 15 Ma. An attempt was made to take a minimum of periapical films covering the right and left maxillary quadrants, the anterior maxilla, the right and left mandibular quadrants, and the anterior mandibular region of each patient. Bitewing roentgenograms were taken whenever possible.

*The subjects observed in this study were patients at the Fernald State School, Waverly, Massachusetts.

†Demerol alone and with Seconal, 1½ grains, was given orally one hour before roentgenographic examination.

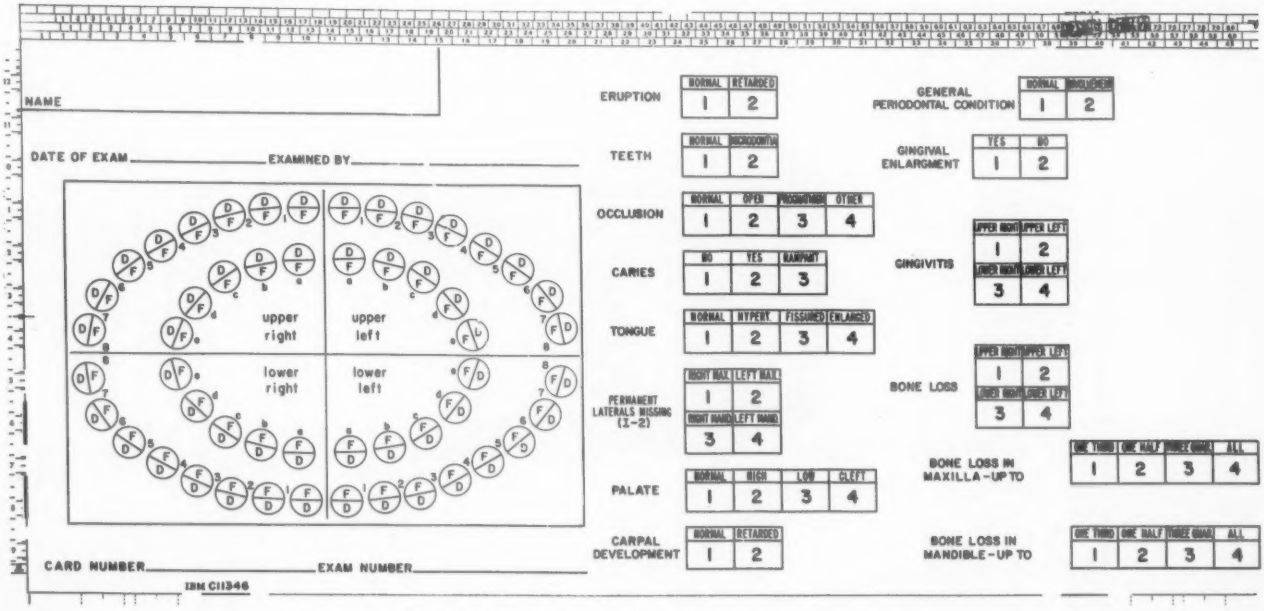


Fig. 2.—I. B. M. card for recording oral conditions in mongolism.

*Histologic Examination.*—Twenty gingival biopsy specimens were obtained for microscopic examination. These specimens were obtained from twenty patients selected to represent various age groups from the total group of mongoloid patients in the study. The tissues were removed from either the maxillary or the mandibular central incisor areas, including the marginal and attached gingiva but avoiding the interdental papillae. Local infiltration anesthesia was employed. Healing was uneventful. Tissues were stained with hematoxylin, eosin, Mallory's connective tissue stain, and periodic acid—Schiff stain for mucopolysaccharides.

The following criteria were employed in evaluating periodontal disease:

1. *Gingivitis:* Gingival inflammation around one or more teeth, manifested by alteration of contour, loss of stippling, redness, hemorrhage, and change in consistency.

2. *Periodontitis:* Inflammation of the marginal or attached gingiva with loss of alveolar crestal bone, observed roentgenographically, and pathologic deepening of the gingival sulcus (pocket formation) with or without exudation from the pocket site.

3. *Periodontosis:* Mobility, migration, and elongation of the maxillary and mandibular incisors with gingival inflammation, pocket formation and suppuration, thickening of the periodontal space, absence or haziness of the lamina dura, destruction of the interdental septa of the alveolar bone in a vertical direction, and alteration of the trabecular pattern of the alveolar bone with enlarged cancellous spaces.

## CLINICAL FINDINGS

The most significant clinical finding in this study was the prevalence of periodontal disease. This condition was observed in ninety six of the 100 patients examined. It was usually characterized by generalized chronic marginal

TABLE I. AGE DISTRIBUTION OF PATIENTS

| AGE GROUP | NO. OF PATIENTS |
|---|---|
| 0— 2 | 4 |
| 3— 6 | 14 |
| 7—12 | 32 |
| 13—17 | 25 |
| 17—30 | 25 |

gingivitis, gingival enlargement, materia alba, stain, calculus, gingival recession and tooth mobility, and pocket formation. Only four patients were free of gingival disease, three of these were under 3 years of age; the fourth was an 18-year-old girl whose roentgenograms showed both vertical and horizontal alveolar bone loss in all quadrants of the mouth.

For simplification in evaluating and comparing the extent and degree of periodontal disease, the patients were divided into groups according to chronological age (Table I).

In Group I (ages 0 to 2) the gingivae around the erupted teeth appeared red, edematous, and spongy and bled easily (Fig. 3). This periodontal condition was classified as marginal gingivitis.

The patients in Group II (ages 3 to 6) presented a more generalized marginal gingivitis and gingival enlargement with materia alba, stain, calculus, gingival recession, and pocket formation (Fig. 4, *A*). Acute necrotizing gingivitis involving the mandibular incisor region was seen in four patients. In four cases the mandibular primary central incisors were peg shaped and had a diastema between them. Supragingival calculus on the lingual surfaces of the incisors and gingival recession were observed.

Fig. 3.—Mongoloid girl, aged 1 year. Note marginal gingivitis in relation to maxillary central incisors and gingivitis in area between peg-shaped mandibular incisors. Note also diastema between mandibular incisors.

In Group III (ages 7 to 12), in which the patients had a mixed dentition, a more severe type of periodontal involvement was evident than was observed in Groups I and II (Fig. 4, *B*, *C*, and *D*). Subacute necrotizing gingivitis was present in twelve patients. Two patients had already lost the permanent mandibular central incisors as a result of periodontal disease.

In the early adolescent and adolescent mongoloids of Group IV (ages 13 to 17), periodontal disease was more advanced than in the younger patients. Gingival recession and pocket formation occurred more frequently and with greater severity. Eight patients in this group had necrotizing gingivitis, and six patients had lost mandibular incisors because of periodontal disease (Fig. 4, *E* and *F*).

All of the older mongoloid patients who made up Group V (ages 17 to 30) had severe periodontal disease which was characterized by gingival recession, gingival detachment, horizontal and vertical bone loss, pocket formation with suppuration, bifurcation and trifurcation involvement in the molar area, and marked mobility of the posterior and anterior teeth. Necrotizing gingivitis was seen in five cases. Four patients in this group lost their lower central incisors.

ROENTGENOGRAPHIC FINDINGS

In assessing the degree of bone loss, the available roentgenograms from each segment of the mouth were evaluated and the amount of bone loss was recorded. The degrees of bone loss were graded arbitrarily from 0 to 4. Grade 4 denoted bone loss approximating three-fourths or more of the root end, while Grade 3 denoted bone loss involving one-half the root surface. Bone loss designated as Grade 2 involved one-fourth of the root surface, and loss of crestal bone was assigned to Grade 1 (Fig. 5).

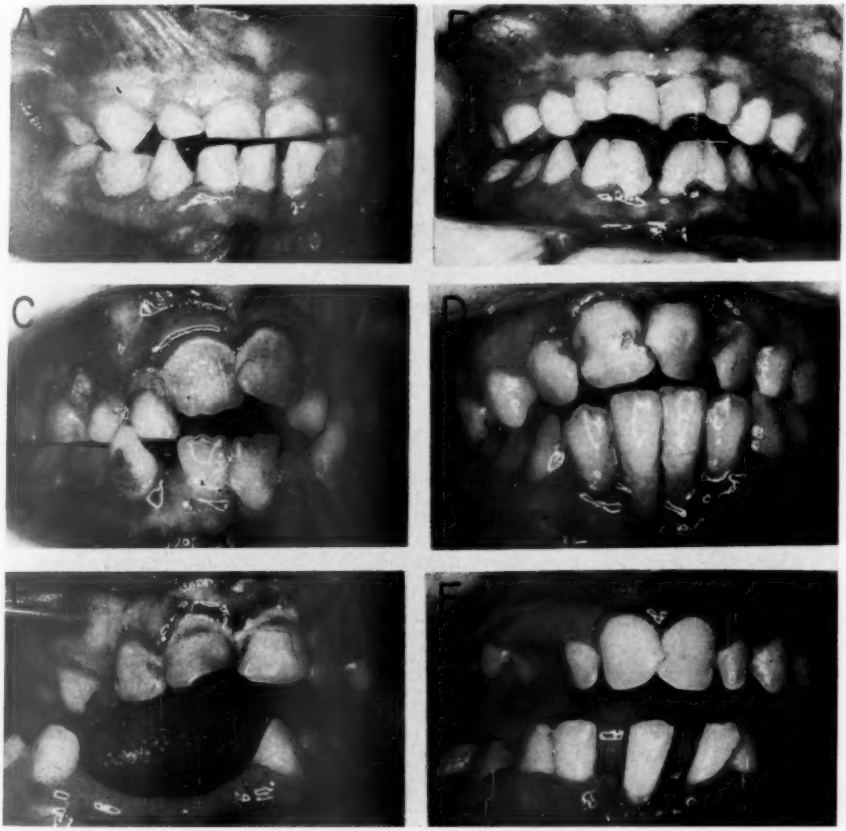


Fig. 4.—*A*, Mongoloid boy, aged 6 years. Note generalized gingival inflammation and hyperplasia, particularly in relation to primary mandibular central incisor area.

*B*, Mongoloid boy, aged 8 years. Note marginal gingivitis with enlargement in mandibular permanent incisor region. Note also diastema between mandibular incisors, as well as materia alba, calculus, and gingival recession.

*C*, Mongoloid boy, aged 8 years. Note severe gingivitis with necrosis (acute necrotizing gingivitis) in maxillary right molar region.

*D*, Mongoloid boy, aged 12 years. Severe generalized periodontal disease with notable recession in mandibular incisor region. Severe bone loss radiographically.

*E*, Mongoloid girl, aged 13 years. Note severe periodontal involvement, which has resulted in loss of mandibular anterior teeth.

*F*, Mongoloid boy, aged 14 years, with generalized periodontal involvement. Left central incisor has been lost through periodontal involvement and right incisor presents a Class III mobility.

In the anterior segment of the mouth bone loss was interpreted from forty-eight roentgenograms of the maxilla and fifty roentgenograms of the mandible (Fig. 6). Bone loss was seen in all forty-eight roentgenograms of the maxillary region and in forty-nine out of fifty roentgenograms of the mandibular area.

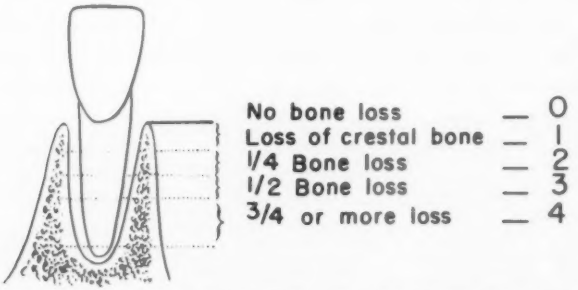


Fig. 5.—Estimation of bone loss from radiographs. By interpretation of radiograph, the amount of bone loss is determined from the assumed normal bone level.

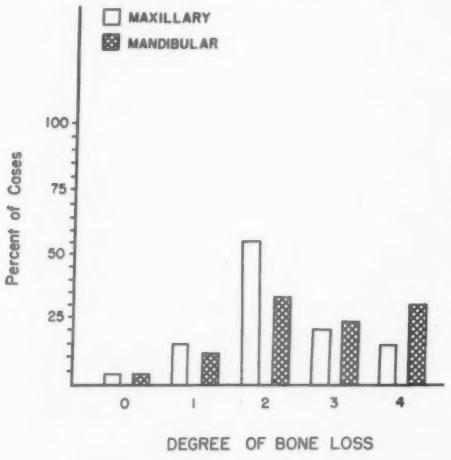


Fig. 6.

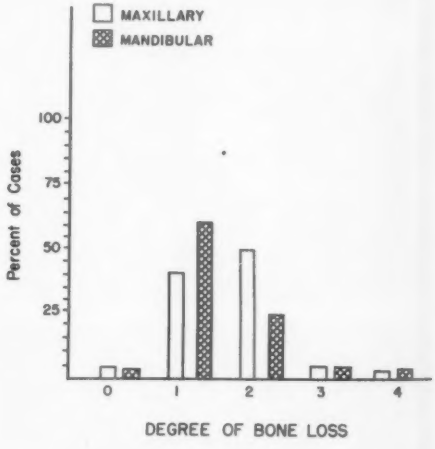


Fig. 7.

Fig. 6.—Degree of alveolar bone loss in relation to maxillary and mandibular anterior teeth of mongoloid patients.

Fig. 7.—Degree of alveolar bone loss in relation to maxillary and mandibular posterior teeth of mongoloid patients.

Fifty-four per cent of the roentgenograms of the maxilla and 32 per cent of those of the mandible showed bone loss approximating one-fourth of the root length. Thirty per cent of the films of anterior mandibular region showed bone loss approximating three-fourths of the root length (Grade 4), while in the anterior maxillary region bone loss was evident in 12.5 per cent of the cases.

In the posterior quadrants the maxillary roentgenograms showed bone loss in fifty-eight out of sixty-one cases. The bone loss around the mandibular teeth was apparent in fifty-five out of fifty-seven roentgenograms studied. The degree

of bone loss is shown in Fig. 7. The most frequent sites of bone loss were the maxillary and mandibular anterior regions. It was more severe in the anterior segments than in the posterior segments, but the incidence was about the same (Figs. 8 and 9).

Fig. 8.

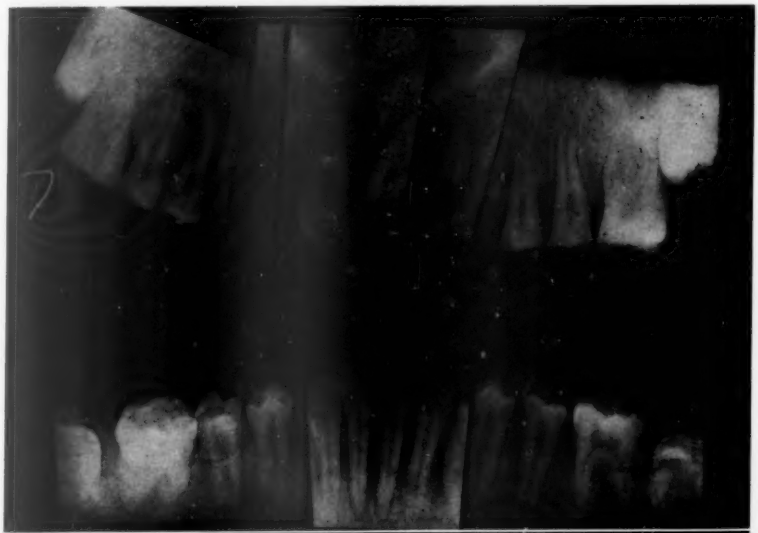

Fig. 9.

Fig. 8.—Roentgenographic survey of case shown in Fig. 4, *D*. Note severe horizontal bone loss in both maxillary and mandibular incisor areas. Posterior areas are relatively free of bone resorption.

Fig. 9.—Roentgenographic survey of mouth of 13-year-old mongoloid boy. In addition to severe anterior bone loss, note vertical bone resorption in maxillary right molar, often seen in mongolism.

The roentgenograms of the mongoloid group were compared with a series of roentgenograms previously taken of a group of mentally defective nonmongoloid patients from the same institution. These two groups were compared with 100 dental school clinic patients of comparable age for whom dental x-rays were available. The proportion of alveolar bone loss for each group is shown in Fig. 10. Ninety-six per cent of the mongoloid patients manifested varying degrees of alveolar bone loss, as compared to 40 per cent of the nonmongoloid group. Only 6 per cent of dental clinic patients showed evidence of bone loss.

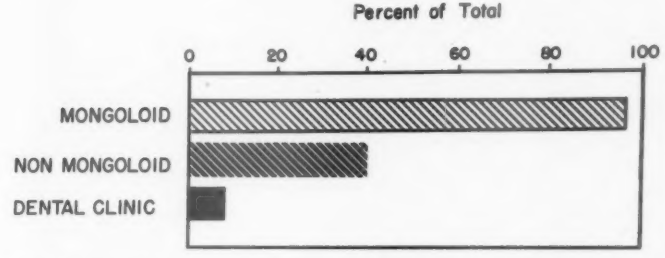


Fig. 10.—Proportion of alveolar bone loss in mongoloid, nonmongoloid, and dental school clinic patients.

MICROSCOPIC FINDINGS

Microscopic examination of the tissue revealed varying degrees of chronic inflammation and epithelial hyperplasia. The epithelium generally presented some parakeratosis and extension of rete pegs. The underlying connective tissue was infiltrated with varying densities of lymphocytes, plasma cells, histiocytes, and in some cases scattered polymorphonuclear leukocytes (Fig. 11, *A*). In several cases there was evidence of epithelial hyperplasia characterized by notable extension of rete pegs (Fig. 11, *B* and *C*). In five cases there was a striking picture of increased vascularity (Fig. 11, *D*). In these cases there was some degree of gingival enlargement clinically and, in addition, there was obvious redness. The blood vessels appeared close to the epithelium, which tended to be rather thin and atrophic (Fig. 11, *D*). These histologic changes are indicative of nonspecific chronic inflammation. The findings with special stains, including Mallory's connective tissue stain and periodic acid-Schiff stain for mucopolysaccharides, were again nonspecific in nature and characteristic of chronic inflammation.

Through the courtesy of Dr. Clemens Benda, Director of Research and Clinical Psychiatry, Walter E. Fernald State School, Waverly, Massachusetts, we were able to study one case at autopsy, that of an 11-year-old mongoloid boy. The mandible and the maxilla were fixed in 10 per cent formalin, decalcified with 5 per cent nitric acid, and embedded in paraffin. Microscopic examination revealed degeneration of osteoblasts and cementoblasts and a diminution in the number of periodontal fibers (Fig. 12). Although no definitive conclusions can

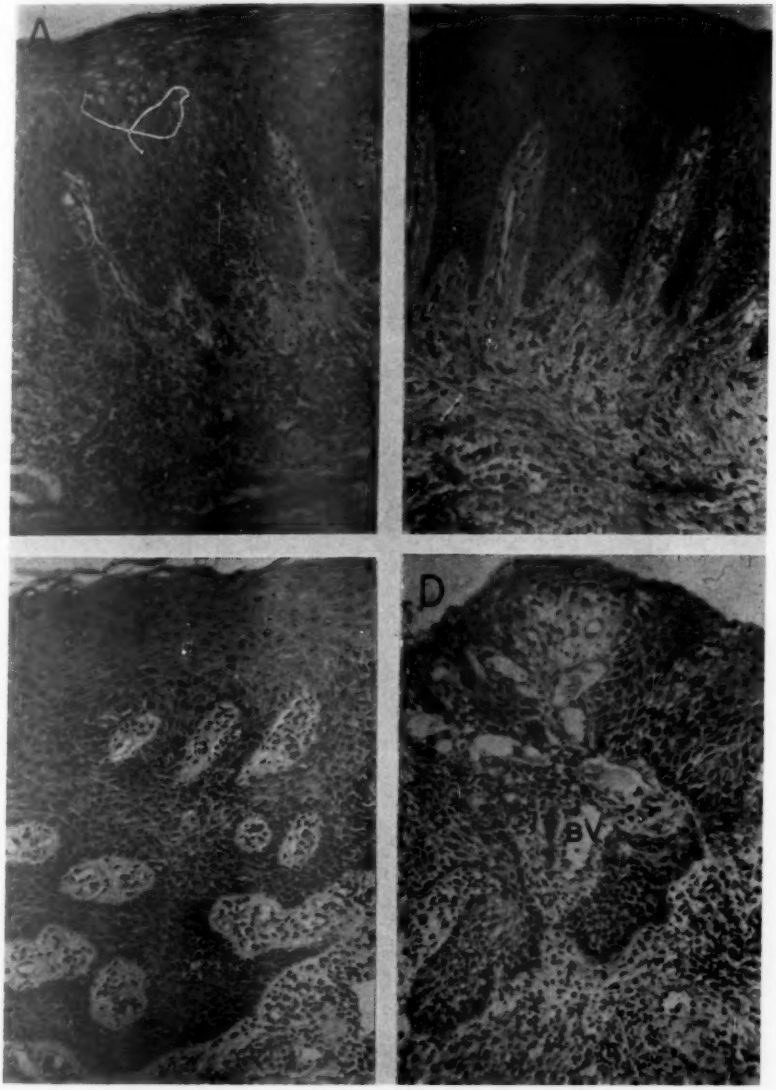


Fig. 11.—Representative biopsy specimens taken from mongoloid patients aged 9 to 12 years. Area between right central and lateral incisors. Low-power magnification of attached gingiva.

*A*, Note edema in stratum spinosum of epithelium and dense inflammatory infiltrate in underlying connective tissue. Cells are primarily lymphocytes, plasma cells, and histiocytes.

*B*, Parakeratosis and relative absence of inflammatory infiltration.

*C*, Epithelial hyperplasia characterized by extension of rete pegs. Moderate inflammatory infiltration.

*D*, Chronic inflammatory infiltration and notable vascularity. Numerous small blood vessels *(BV)* are seen beneath epithelium, which is thin and atrophic in areas.

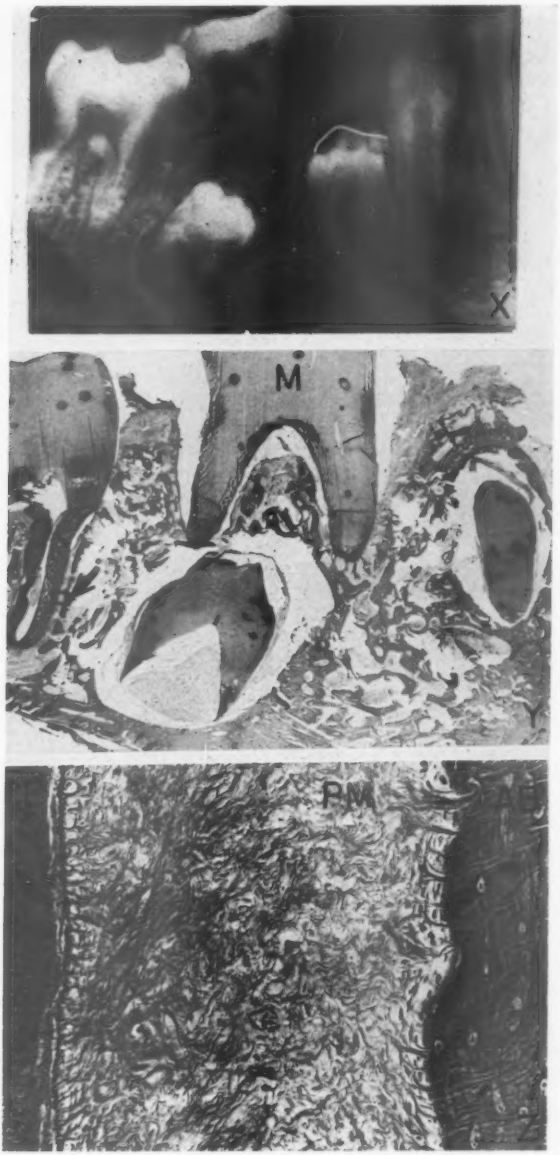


Fig. 12.—*X*, Autopsy specimen of 11-year-old mongoloid boy. Radiograph of mandibular right molar area.

*Y*, Survey section of area shown in *X*. Note primary molar *(M)* which is undergoing exfoliation. Arrow points to area of periodontal membrane mesial to first permanent molar.

*Z*, High-power study of area outlined in *X* on mesial surface of first molar. Note degeneration of cementoblasts in relation to cementum *(CE)* and osteoblasts in relation to alveolar bone *(AB)*. Fibers of periodontal membrane *(PM)* appear reduced in number for this age group, and their arrangement is somewhat abnormal.

be drawn from a single autopsy, the findings are suggestive of an underlying systemic alteration related to connective tissue and described in the literature as being characteristic of periodontosis.

## DISCUSSION

Many conditions, such as advanced maternal age,[53-55] hemorrhage, threatened abortion,[56] pathologic abnormalities of the uterus and placenta,[57] acute intercurrent infections early in pregnancy,[58] psychophysiologic states[59] (emotional and physical stress), and other environmental influences, have been implicated as etiological factors in mongolism. Recently it has been suggested that the mongoloid child is the result of a chromosomal aberration (an extra chromosome).[1] According to a current report by Toolan,[60] hamsters injected at birth with fractions of cell-free filtrates of transplanted human tumor cells, as well as certain tissues derived from human beings and rats carrying spontaneous cancer, have developed a mongoloid-like deformity. These mongoloid-like animals probably represent a phenocopy of the mongoloid and are characterized by fragile bone structure with paper-thin skulls, protruding eyes and tongue, and abnormalities of the teeth and jaws.

Although a host of factors have been implicated in the etiology of periodontal disease, no characteristic bacterial flora or specific inflammatory change in the gingiva and alveolar bone loss has been observed in patients with this disturbance. In the present study, periodontal disease was observed in 96 per cent of the patients examined.

*Local Factors.*—Some of the local factors that play an important role in the causation of periodontal disease in the mongoloid are lack of dental care, lack of oral hygiene (toothbrushing and interdental stimulation), lack of routine prophylactic treatment (scaling and curettage), absence of functional stimulation derived from mastication of food, food impaction, severe gingival disease in the very young mongoloid child, untreated necrotizing gingivitis, supragingival and subgingival calculus and materia alba, enlarged tongue (macroglossia), severe malocclusion associated with mongolism, such habits as bruxism or abnormal pressure of the tongue against the teeth, and tooth-clenching.

Although local factors definitely contributed to the periodontal disease in these patients, the severe alveolar bone loss and the intense inflammatory changes observed clinically and microscopically in the gingiva suggest a relationship between brain damage, systemic factors, and the periodontium.

*Systemic Factors.*—Magendie[61] and Samuel[62] first postulated the theory of the trophic function of the nervous system. A relationship between the central nervous system and various gastric and oral lesions has been observed for many years and has led to the report by Speransky[63, 64] of a general theory of nervous control of disease processes.

In general terms, the theory states that neurodystrophic processes enter the composition of all pathologic processes. Experiments carried out by Speransky's associates, Skoblo[65] and Pigalew,[66] who placed glass spheres and semicircular rings in the area of the hypothalamus to produce mild chronic irritation of the

central nervous tissue in dogs, produced periodontal disease which began as edema and hemorrhage and progressed to ulceration. Ulcers were also found on the oral mucosa. Alterations of the teeth were also noted, as were extensive calculus deposits. The teeth became soft and crumbled under masticatory stresses. Entin[67, 68] added to these observations and noted the formation of a condition similar to alveolar pyorrhea with calculus deposition, tooth mobility, and gingival inflammation and recession. Schachter and Schachter[69] confirmed many of these experimental findings and described bleeding and pus formation at the gingival margins, discoloration of teeth, loss of teeth, and gastrointestinal ulceration. Quintarelli and Chauncey[70] have recently enlarged these findings and have reported initial oral changes consisting of gingival edema and gingival and mucosal hemorrhage progressing to ulceration. Periodontal pocket formation with suppuration was noted, and infrabony pockets and bone loss were studied microscopically. Acute necrotizing gingivitis was found superimposed on chronic marginal gingivitis in several animals.

These experimental findings suggest a possible mechanism for the severe periodontal disease and the occurrence of necrotizing gingivitis in 29 per cent of the patients. The brain pathology may in some way alter the resistance of the periodontal tissues to local irritation.

The prevalence of the severity of bone loss in the region of the mandibular incisors requires further histologic study. The constant occurrence of alveolar bone loss in this area in the young mongoloid patient is again suggestive of a systemic factor or an inborn error in normal maturation of alveolar bone.

## SUMMARY AND CONCLUSIONS

In a group of 100 mongoloid patients, 1 to 30 years of age, severe periodontal disease was found in 96 per cent.

The occurrence of superimposed necrotizing gingivitis in twenty-nine patients requires further study.

Although local factors were present in abundance, the severe alveolar bone loss and the intense inflammatory changes in the gingiva suggest a relationship between systemic factors, brain damage, and the periodontium.

Although it is impossible to conduct controlled experimental studies on normal growing children, either with or without the precision practicable in the laboratory, productive studies are possible on mongoloid children. In mongoloids periodontal disease is found almost universally from early childhood through adulthood. This affords the dental investigator an opportunity to observe periodontal disease early in the patient's life and to follow its progress. The role of local and systemic factors may be evaluated independently, and the relationship between local and systemic factors will thus be more clearly understood.

The authors are grateful to Dr. Harry C. Solomon, Commissioner, Department of Mental Health in Massachusetts, Dr. Malcome J. Farrell, Director of the Walter E. Fernald State School in Waverly, Massachusetts, Dr. Clemens E. Benda, Director of Research and

Clinical Psychiatry, Walter E. Fernald State School, Waverly, Massachusetts, and Dr. Richard McDonough, Resident Dentist at the Walter E. Fernald State School, Waverly, Massachusetts, for their interest and cooperation in making patients available for this study.

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## Section by the

## ARMED FORCES AND PUBLIC HEALTH SERVICE

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# PRIMARY MALIGNANT MELANOMAS OF THE PAROTID GLAND

*George W. Greene, Jr., D.D.S.,* Washington, D. C., and*
*Joseph L. Bernier, Colonel, DC, USA***

PRIMARY malignant melanomas of the parotid gland have been reported in comprehensive studies of parotid tumors[1, 2] and in isolated case reports.[3, 4] Criteria for the establishment of the tumor as primary in the parotid gland have not been clearly defined.

The material at the Armed Forces Institute of Pathology was reviewed to shed further light on this lesion. The melanoblast is a normal and constant constituent of oral epithelium, as shown by Laidlaw and Cahn.[5] The development of the parotid gland begins in the fourth week of embryonic life with a downgrowth of oral epithelium, as solid buds with club-shaped ends, into the underlying mesenchyme.[6] It was reasoned, therefore, that these epithelial buds would contain melanoblasts which would become a part of the parotid gland. The presence of these cells in the human parotid gland has not been demonstrated heretofore; however, in this study normal human parotid gland tissue prepared by the freeze-dry technique and stained by the dopa method was positive for melanin (Fig. 1). In addition, dopa stains were done on normal parotid gland tissue of dogs, and positive reactions were obtained in some of the cells (Fig. 2). It would appear, therefore, that such dopa-positive cells are present in human parotid gland tissue.

Malignant melanomas most commonly arise in the skin, but some cases in mucous membrane have been reported.[7, 8] In both instances these tumors arise in a junctional nevus per se or in the junctional component of a compound nevus. A nevus has never been reported in the parotid gland. Hence,

From the Armed Forces Institute of Pathology and the Central Laboratory for Pathologic Anatomy and Research, Veterans Administration.
*Chief, Environmental Oral Pathology Branch, Armed Forces Institute of Pathology.
**Chief, Dental and Oral Pathology Division, Armed Forces Institute of Pathology.

melanoma in the parotid gland is considered to arise directly from melanoblasts in the tissue. This situation would be considered comparable to that of malignant melanotic tumors that arise in the eye.[9-11]

The files at the Armed Forces Institute of Pathology revealed five cases that had been diagnosed as malignant melanoma of the parotid gland. In order to determine whether the lesions were primary at this site, the following criteria were established: (1) the presence of dopa-positive cells in normal parotid gland tissue, (2) demonstration of malignant melanomas in the parotid gland, and (3) inability to demonstrate any other primary site. The first requisite, the presence of dopa-positive cells, could not be substantiated in our five cases, since the material on all the cases was fixed in formalin. In

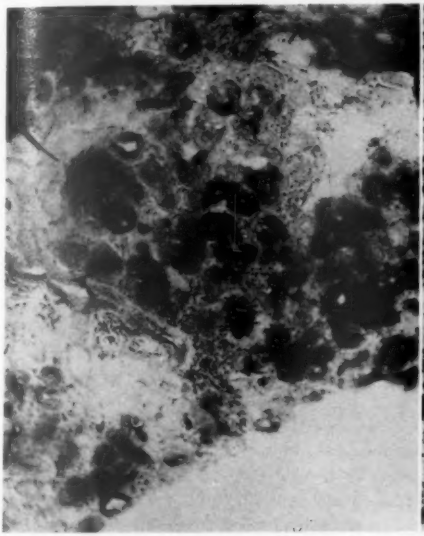

Fig. 1.

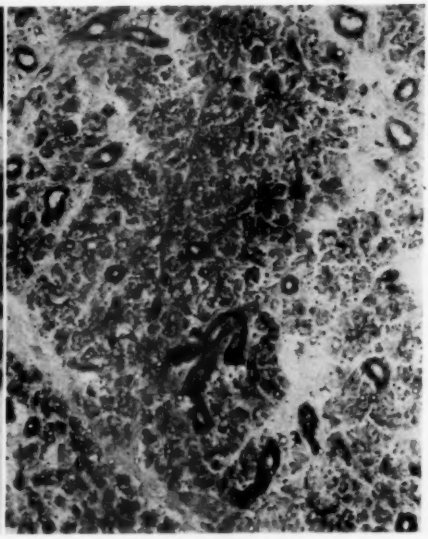

Fig. 2.

Fig. 1.—Dopa-positive material is present in some of the ductal and acinar cells of the human parotid gland. AFIP negative No. 59-7106. (Dopa stain. Magnification, ×305; reduced ¼.)

Fig. 2.—Dopa-positive material is evident in ductal and acinar cells of the parotid gland of the dog. AFIP negative No. 59-7104. (Dopa stain. Magnification, ×115; reduced ¼.)

all our cases, however, the presence of melanin-containing cells was demonstrated by the ferrous ion uptake stain of Lillie,[12] which is considered a specific reaction for melanin. The remaining two criteria will be demonstrated in the case reports. Several cases of melanoma, metastatic to the parotid gland, were reviewed and were distinguished from primary lesions either by a history of another primary site or by histologic appearance. Metastatic lesions appeared either in intraparotid lymph nodes or as well-delineated lesions when noted in parotid gland tissue, whereas the primary lesions were infiltrative and poorly demarcated or completely replaced the parotid gland tissue.

CASE REPORTS

CASE 1 (AFIP ACC. NO. 6723).—A 23-year-old white man was admitted to the hospital with a diagnosis of chronic peritonitis. History revealed that the patient had undergone an operation on the left parotid gland two months earlier, at which time a diagnosis of melanoma was made. The patient died the day following his admission.

*Autopsy Findings.*—The left parotid gland was enlarged, nodular, and pigmented. Numerous tumors, varying in consistency and ranging in diameter from 2 mm. to 2.5 cm., were irregularly distributed in the subcutaneous tissues of the chest, back, abdomen, arms, and legs. These nodules showed pigmentation, and many appeared quite blue in color.

The peritoneum, liver, kidneys, gastrointestinal tract, pleura, heart, and lungs were studded with numerous small pigmented tumors ranging from a few millimeters to a centimeter in diameter.

Microscopically, the tumor was noted in all areas in which it was noted grossly. The parotid gland was replaced by melanoma cells arranged in an alveolar pattern. The cells were round or oval with well-defined borders. The cytoplasm was pink, and in several areas it was noted to contain melanin pigment. The melanin pigment was more pronounced in the metastatic nodules (for instance, those in the liver). The tumor was diagnosed as malignant melanoma of the parotid gland. No evidence of eye changes or skin lesions was recorded in the patient's records.

CASE 2 (AFIP ACC. NO. 491198).—A 67-year-old white man had a tumor in the right parotid gland for two or three years. Recently it had increased rapidly in size. The general physical examination was essentially normal.

The tumor, which was removed in May, 1951, measured 4.5 by 3.5 by 0.5 cm. On sectioning, one-half of the lesion appeared to be normal salivary gland tissue, while the other portion contained a rounded, apparently encapsulated swelling. This swelling was firm and rubbery in consistency, and the cut surface was gray and reddish brown in color. The cut surface did not show the same degree of encapsulation as was expected on the basis of the external examination.

The section consisted of aggregates of melanoma cells. The cells were round or oval in shape, and the cytoplasm was pink and vacuolated in appearance (Fig. 3, top and lower left). Many of the cells contained yellowish brown granules of pigment (Fig. 3, top and lower right) that was positive with Fontana's stain and Lillie's ferrous ion uptake. The tumor was diagnosed as primary malignant melanoma of the parotid. The patient died in May, 1952, one year after surgery. Death was apparently due to cerebral metastasis, although an autopsy was not performed.

CASE 3 (AFIP ACC. NO. 553615).—A 28-year-old white man had a small tumor of the left parotid gland removed in November, 1949. The tumor was diagnosed as undifferentiated adenocarcinoma of the parotid gland. In June, 1950, the patient was hospitalized because of a diffuse swelling in the area of the excision. The opinion of the Tumor Board was that the mass represented scarring at the operative site and that no treatment was indicated. In May, 1952, the patient was hospitalized with a diagnosis of splenomegaly of undetermined origin. A laparotomy was performed, and the left kidney and adrenal gland were removed with a large tumor mass. This was diagnosed as metastatic malignant melanoma. The slides of the parotid tumor were reviewed, and this lesion was also diagnosed as malignant melanoma. In March, 1953, the patient was again hospitalized because of the tumor mass in the left parotid gland. A total parotidectomy was done, and the tumor was diagnosed as malignant melanoma.

The patient was again hospitalized in June, 1953, with evidence of widespread metastases, and he died in July, 1953.

The histologic features of the metastatic foci were essentially similar to those of the original tumor of the parotid gland. The tumor cells varied from oval to round, while in some areas they were spindle-shaped. The cytoplasm was eosinophilic, and in many instances it contained yellowish brown pigment granules. Fontana's stain, Lillie's ferrous ion uptake, and melanin bleach all produced positive results. The tumor was diagnosed as malignant melanoma of the parotid gland.

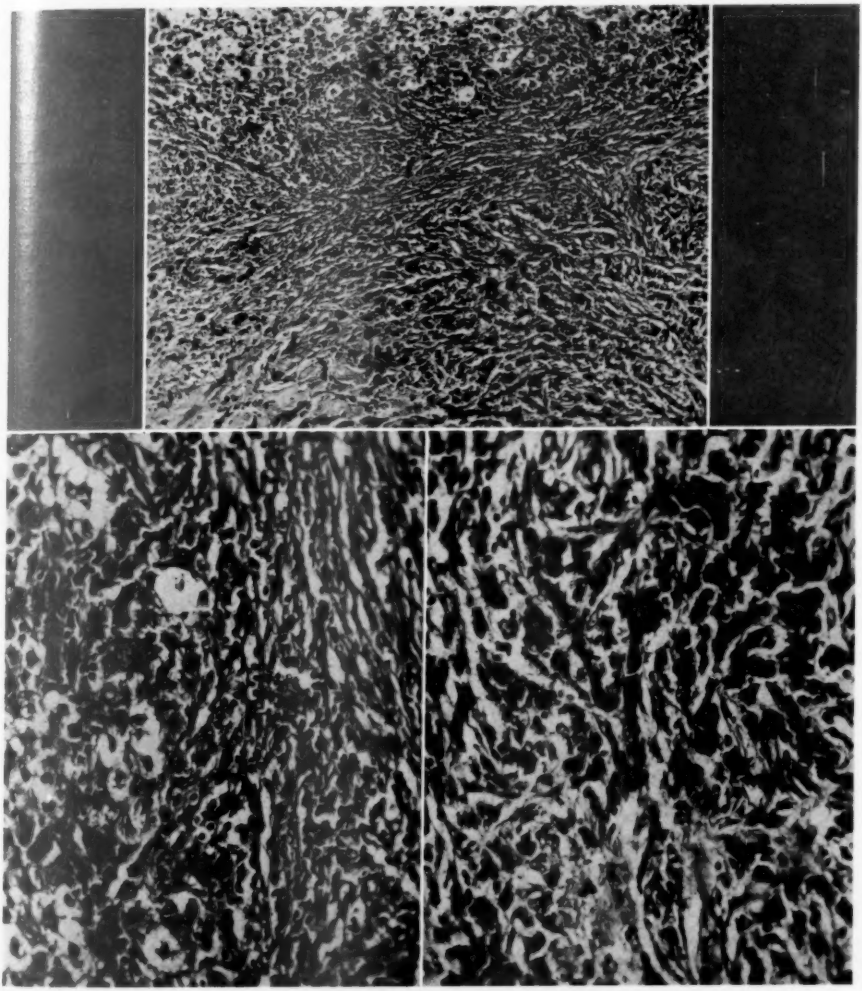

Fig. 3.—Case 2. *Top:* The parotid gland is replaced by melanoma cells. Some show vacuolated cytoplasm *(A)* and others contain granules of melanin pigment *(B)*. AFIP negative No. 59-7101. (Hematoxylin and eosin stain. Magnification, ×115; reduced ¼.)

*Lower left:* High-power view of boxed area *A*, showing round and oval cells with vacuolated cytoplasm. AFIP negative No. 59-7103. (Hematoxylin and eosin stain. Magnification, ×305; reduced ¼.)

*Lower right:* High-power view of boxed area *B*, showing cells heavily laden with melanin pigment granules. AFIP negative No. 50-7102. (Hematoxylin and eosin stain. Magnification, ×305; reduced ¼.)

CASE 4 (AFIP ACC. NO. 598946).—A 41-year-old white man underwent a partial parotidectomy for a mass in his left parotid gland in December, 1952. The lesion was diagnosed as inflammation and hyperplasia of the parotid gland. Two months later the patient noticed a painful mass in the gland. The mass was aspirated, and a gray, friable, necrotic tissue was found. The tissue was diagnosed as an undifferentiated epithelial tumor of salivary gland origin. The patient was returned from Japan and in July, 1953, a lymph node of the neck was biopsied. Histologic findings were negative, as were the chest film and other tests taken at this time.

Several days later the left parotid area was explored, and a large cystic mass measuring 4 cm. in diameter was noted in the infratemporal area beneath the parotid gland. The cyst appeared encapsulated and was composed of soft, caseous, necrotic material, dark reddish gray in color. The mass was removed in toto, and it did not involve the remaining parotid gland tissue, which was left intact.

Histologic examination revealed a compact mass of undifferentiated epithelial cells. In some areas the cells formed a syncytial mass of eosinophilic cytoplasm in which the nuclei were round or oval and each had a prominent nucleolus. In other areas the cells were grouped in clusters and separated by connective-tissue septa. These cells had distinct outlines, eosinophilic or vesicular cytoplasm, round or oval nuclei, and prominent nucleoli. In both areas many of the cells contained yellowish brown granules and did not stain with Prussian blue but did with Fontana's and Lillie's ferrous ion uptake stains. The lesion was diagnosed as malignant melanoma of the parotid gland.

CASE 5 (AFIP ACC. NO. 605627).—A 66-year-old white woman had noted a lump in the parotid region for eight weeks. The lesion was not painful, and the overlying cutaneous tissue exhibited no significant changes. The lesion was identified as a "tumor of the parotid gland," both preoperatively and postoperatively. Upon removal, the parotid lesion was noted to be indurated, solid, and darkly pigmented, as was one of the superior anterior

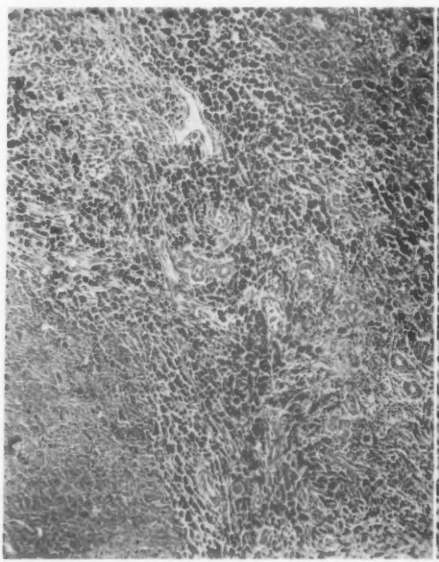

Fig. 4.

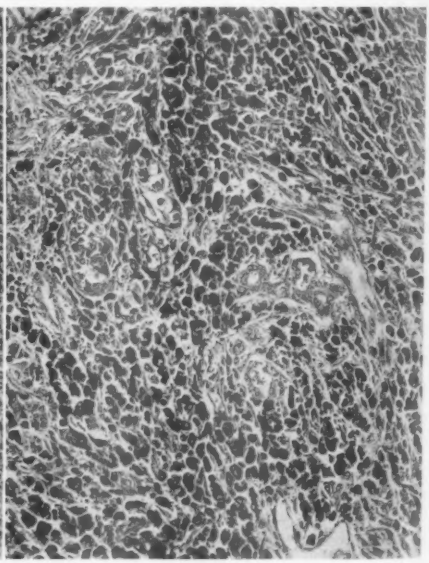

Fig. 5.

Fig. 4.—Case 5. Melanoma cells containing pigment replacing parotid gland tissue. AFIP negative No. 59-7089. (Hematoxylin and eosin stain. Magnification, ×80; reduced ¼.)

Fig. 5.—Case 5. Higher-power view of section shown in Fig. 4. Note pigment-containing cells and remnants of parotid gland ducts. AFIP negative No. 59-7094. (Hematoxylin and eosin stain. Magnification, ×145; reduced ¼.)

cervical lymph nodes. Subsequently, a complete, careful re-examination of the patient was performed; there were no positive findings, and there was no previous history of a skin lesion or "mole."

Fig. 6.

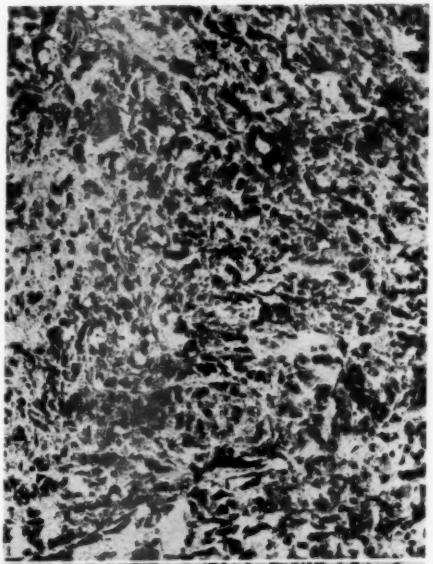

Fig. 7.

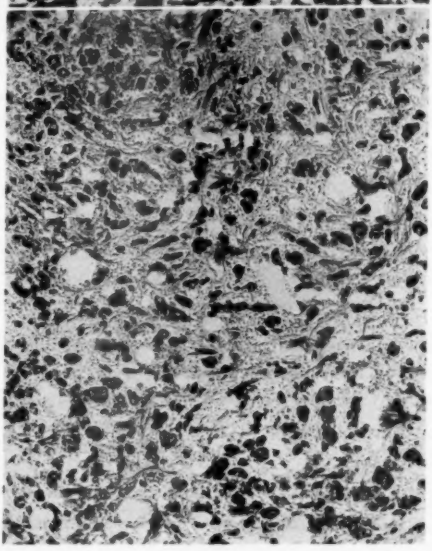

Fig. 6.—Case 5. Pigment granules are positive with Fontana's stain. AFIP negative No. 59-7098. (Magnification, ×145; reduced ¼.)

Fig. 7.—Case 5. Pigment granules are positive. AFIP negative No. 59-7097. (Lillie's ferrous ion uptake stain. Magnification, ×145; reduced ¼.)

Histologic examination revealed a richly cellular tissue composed of anaplastic "nevus cells." The cells varied in size, shape, and staining quality, and several mitotic figures were observed. These cells contained variable amounts of yellowish brown pigment granules (Figs. 4 and 5). The Prussian blue stain produced negative results, Fontana's stain (Fig. 6) and Lillie's ferrous ion uptake stain (Fig. 7) gave positive results, and the melanin bleach removed all the pigment present in the slide (Fig. 8). The lesion was diagnosed as malignant melanoma of the parotid gland.

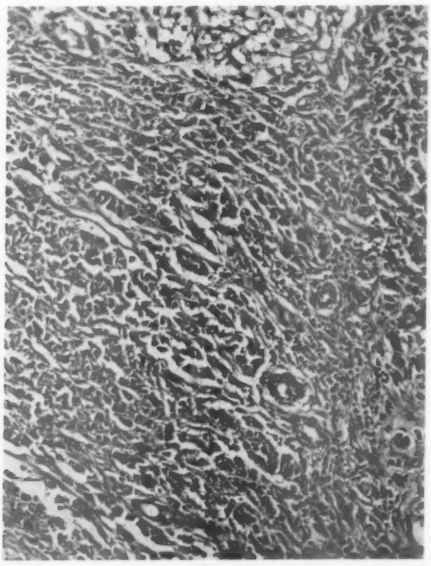

Fig. 8.—Case 5. Pigment was completely removed from melanoma cells. AFIP Negative No. 59-7095. (Melanin bleach. Magnification, ×145; reduced ¼.)

DISCUSSION

The question of whether a malignant melanoma is primary in the parotid gland is admittedly a controversial one. Until recent years, however, even a diagnosis of primary malignant melanoma of mucous membrane was accepted with a certain amount of skepticism. Several recent articles[8, 13-15] have reported primary malignant melanomas in various locations on mucous membrane. Greene and associates[13] reported five cases of primary malignant melanoma on oral mucous membrane, and the criteria used were (1) demonstration of malignant melanomas in the oral mucosa, (2) presence of intraepidermal (junctional) activity, and (3) inability to demonstrate any other primary site. The most important criterion is the demonstration of junctional activity in lesions on mucous membrane and skin, but this is not applicable in malignant melanomas of the parotid gland, since these tumors arise directly from melanoblasts present in the parotid tissue. Therefore, certain changes were necessary in the standards for establishment of malignant melanomas as

primary in the parotid gland. The criteria considered acceptable were (1) presence of dopa-positive cells in the parotid gland, (2) demonstration of malignant melanoma in the parotid gland, and (3) inability to demonstrate any other primary site.

In our five cases the criteria demonstrating the occurrence of malignant melanoma in the parotid gland and the inability to demonstrate any other primary site were fulfilled. From frozen-dried sections of normal parotid gland, in both human beings and dogs, dopa-positive cells were demonstrated. They were considered normal and constant findings in the parotid gland. Several cases of metastatic melanoma of the parotid gland were reviewed but were not included in this study. In our material the tissue was fixed in formalin; hence, the dopa reaction was not effective. In all instances, however, the pigment-containing cells were negative when stained with Prussian blue but positive with Fontana's stain and Lillie's ferrous ion uptake stain, and the melanin bleach procedure was effective in all instances.

In all our cases the lesion was first noted in the parotid gland. There was evidence of recurrence or spread of the lesion from two months to four years after the initial lesion appeared. In all instances in which the tumor spread to other areas, these secondary foci in no way suggested, even when examined histologically, that they might represent a primary focus. In the fatal cases (Cases 1, 2, and 3) there was evidence of widespread metastases; in Cases 4 and 5, even though the lesions appeared encapsulated, there was extension beyond the capsule, as revealed by histologic examination. Following surgical removal, there were no recurrences in a follow-up period of four years in Cases 4 and 5.

The clinical history, histologic examination, and all other factors indicate that the lesions were primary in the parotid gland. In Case 2 the histologic material was reviewed by McDonald,[1] and he agreed that the tumor was a malignant melanoma, primary in the parotid gland, and that it was similar in histologic appearance to the three cases he and his co-workers had reported. Our five cases, together with those previously reported in the literature, should establish malignant melanoma of the parotid gland as primary. The number is so small that it is difficult to interpret the clinical behavior of the tumor; however, it appears to be similar to malignant melanomas found elsewhere in the body. The recognition of primary malignant melanoma of the parotid gland has great clinical significance, since a primary lesion is amenable to appropriate therapeutic procedures whereas a metastatic lesion is not.

## SUMMARY AND CONCLUSIONS

Five cases of primary malignant melanoma of the parotid gland were reported. The presence of melanoblasts or dopa-positive cells was demonstrated in frozen-dried sections of normal human and dog parotid gland tissue. In our five cases the initial lesion was first noted in the parotid gland, and there was no history or evidence of any other primary site.

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# SCLERODERMA

## Its Oral Manifestations

*Jack Traiger, Captain, DC, USA*

It is common knowledge that many systemic diseases manifest themselves in the oral cavity. Scleroderma is one of this large group of diseases.[1]

Scleroderma is characterized by a peculiar hardening and rigidity of the skin. It occurs at any age and affects women and girls more often than men and boys. As a rule, the onset of the disease is gradual and insidious. There are prodromal symptoms of pain and swelling of the joints, especially the hands, resulting in limitation of movement. The disease is often diagnosed as arthritis. Skin involvement is either localized or general. In the first stage edema is the most marked symptom; this is followed by atrophy and finally by an induration which causes a leatherlike condition of the skin. The skin of the face and neck alone is frequently involved. Due to a thin, smooth, glistening, often mottled and pigmented skin directly overlying the bone, the patient has a mummylike appearance. The skin may shine like ivory, and the lines of expression and facial wrinkles are absent.

The lips are thin and rigid, and the cheeks lose their elasticity; this, combined with a decrease in the size of the oral orifice, interferes with eating and speaking.

The oral mucosa may be affected, especially if the skin of the face is involved. The mucosa is thin, pale, and tender, with a tendency to ulcerate. Often there is resorption of the alveolar bone together with tilting of the teeth due to extrinsic pressure. Involvement of the muscles of mastication prevents opening of the jaw; this produces a false ankylosis which adds to the difficulties already mentioned. In fact, limitation of mouth opening may be the initial complaint. Dysphagia associated with esophageal involvement is a frequent finding.

### ETIOLOGY

The cause and mechanism of systemic sclerosis are unknown. The disease is often believed to be caused by neurotrophic factors, although some investigators think that abnormal thyroid function plays a role. Mental strains, overexposure to sunlight or cold, drug intoxications, and acute infections have been cited as etiological factors. Best and Taylor[2] state that it is a systemic disease

From the Oral Surgery Section, Post Dental Clinic, Fort Jay, Governors Island, New York, New York.

involving the collagenous tissues of the body including the collagenous tissues of the blood vessels. An antigen-antibody allergic type of reaction has recently been postulated.[3]

HISTOPATHOLOGY

In well-defined scleroderma the most important pathologic finding is hypertrophy of the collagenous tissue of the skin and subcutaneous tissues. In the early stages there is a rich inflammatory infiltrate which gradually disappears. In the papillae of the corium the fine, delicate collagen fibers are transformed into homogeneous course fibers that intertwine with one another in all directions. This causes a flattening-out of the papillae and the epithelium. The connective-tissue fibers in the deeper layers of the corium are transformed into greatly enlarged coarse bundles pressed closely together. The walls of the blood vessels are thickened, and their lumina are decreased in size.[4]

DISCUSSION

According to Stafne,[5] abnormal widening of the periodontal membrane space occurs in approximately 10 per cent of cases of scleroderma. Stafne goes on to state: "This space is created by a thickening of the periodontal membrane as a result of an increase in the size and number of collagen fibers. The enlarged space is almost uniform in width, surrounds the entire root of the tooth and makes the tooth appear as if it is being extruded rapidly from its socket. Increased size of the periodontal space, however, is created at the expense of the alveolar socket. There is roentgenographic evidence of resorption and destruction of the lamina dura, which are followed by sclerotic changes in the walls as evidenced by the white radiopaque line that eventually outlines the limits of the enlarged socket. The changes that take place seem to have a predilection for the posterior teeth." (Fig. 1.)

CASE REPORT

T. F., a 38-year-old Filipino, an enlisted man in the Army, was admitted to the Fort Jay Hospital on March 28, 1960, with chief complaints of weight loss, swellings of the hands and face, and multiple joint pain. These symptoms had been present for eighteen months and were getting progressively worse. The patient also complained of becoming easily fatigued.

In the spring of 1958 the patient suffered multiple joint pain that was so severe as to be incapacitating; the pain was worse in the shoulders and knees. The patient was hospitalized for a month and treated with red, coated pills (sodium salicylate?) which gave him some relief. He was told that he had arthritis. He did fairly well for about eight months, when he suddenly became impotent. Despite various medications, he had not been able to have intercourse for nine to ten months, as he had been unable to maintain an erection.

In November, 1959, he again began to have difficulty with his joints and noted a swelling of his hands and face. When admitted to Fort Jay Hospital, he felt that his face was still swollen; his hands definitely were, and he was unable to make a fist. Other joints had never swollen, and joint pain was not made worse by activity. He was restless at night due to joint and muscle pains. He had had chills nightly for two weeks. Recently he had

perienced some difficulty in swallowing. Past history revealed that during World War II the atient was a POW in a prison camp in the Philippines and that he had once been hospitalized ecause of malaria. Recently he had developed a cough with production of dark sputum.

Physical examination revealed a short, lethargic Filipino man in no acute distress, who as alert and cooperative. The skin of the face, hands, and fingers was glistening, tight, nd smooth (Fig. 2); it could not be picked up between the fingers. Finger motion was

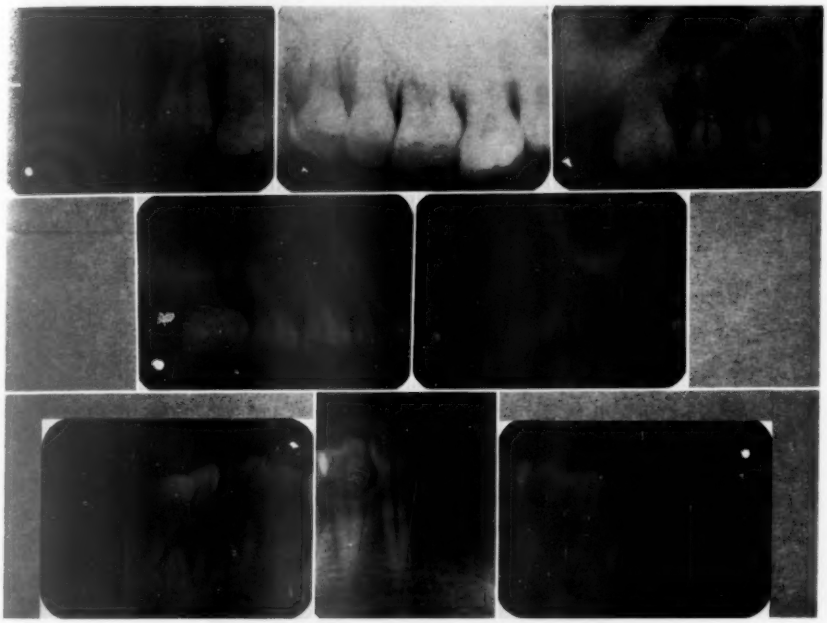

Fig. 1.—Note characteristic thickening of periodontal membrane involving posterior teeth.

Fig. 2.—Typical masklike facies, with glistening, tight, and smooth skin.

limited, and the patient was unable to make a fist (Fig. 3). There was a loss of pigmentation over the upper and midcervical vertebrae as well as a tautness of the skin of the upper extremity and chest.

Because of the rigidity of the tissues, the patient could not open his mouth wide or alter his facial expression to any appreciable degree (Fig. 4).

Fig. 3.—Stiffness of the joints and crippling deformities.

Fig. 4.—Rigidity of tissues, causing limited mouth opening.

Dental roentgenograms revealed a thickening of the periodontal membrane with an increase in the width of the lamina dura.

Neurologic examination revealed some loss of touch of the fingers. Other laboratory findings were unremarkable.

A barium-Micropaque-water mixture was administered orally, and a normal swallowing act was observed flouroscopically. Examination of the esophagus revealed some lack of tone and a decrease in the amount of peristaltic activity. The changes seen in this case were very mild, with just the earliest evidence of scleroderma-like changes of the esophagus.

Examination of the lung fields revealed the presence of diffuse nonspecific fibrotic changes, more marked in the right lung.

There was no significant narrowing of the joint spaces. The distal phalanges were neither demineralized, shortened, or pointed in a peglike fashion as is sometimes seen with changes secondary to scleroderma.

The patient was placed on Potaba (potassium-amino benzoate).[6] No immediate improvement in his condition was noticed.

## CONCLUSION

The most common finding of dental significance in scleroderma is rigidity of the lips and difficulty in retracting them. Pseudoankylosis may be caused by restricted mouth opening, and this may lead to poor oral hygiene and its sequelae. This may also occur when there is sufficient crippling of the hands to prevent toothbrushing. It may be difficult to secure intraoral roentgenograms. Once obtained, they may demonstrate the characteristic findings already discussed. Periodic examination should be made to determine whether these changes are generally permanent or transitory.

In the case just described, routine dental examination while the patient was in the hospital confirmed the diagnosis previously made by the medical service. The patient was transferred to Walter Reed Army Hospital for further medical evaluation pending recommendation for retirement.

## SUMMARY

1. A case of scleroderma has been presented to demonstrate the manifestations of the disease.

2. The characteristic dental roentgenographic finding of widening of the periodontal membrane was observed in the patient.

3. In order to determine whether the changes in the periodontal membrane are permanent or transitory in cases of systemic sclerosis, periodic dental roentgenographic examinations are recommended.

4. The dental profession is again reminded of its obligation to detect systemic diseases when they present themselves in the oral cavity.

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# ANNOUNCEMENTS

## Midwest Society of Periodontology

The Society will hold its third annual meeting on Feb. 5, 1961, at the Blackstone Hotel in Chicago, Illinois. The following papers will be presented:

Correlation of Clinical and Microscopic Pathology. Donald A. Kerr, D.D.S., M.S.
Experimental Surgery: Wound Healing in Periodontics, Frank M. Wentz, D.D.S., M.S., Ph.D.
A Practical Approach to the Treatment of Periodontal Disease. John R. Wilson, A.B., D.D.S.

## International Association for Dental Research

The International Association for Dental Research will hold its thirty-ninth annual meeting at the Statler Hotel in Boston, Massachusetts, March 23 to 26, 1961.

Approximately 300 papers will be delivered, and several symposia are planned for this meeting. The program will include such topics as dental caries (both human and animal), properties of enamel and dentin, wound healing, microbiology, nutrition and metabolism, and salivary studies.

Close to 1,000 persons, including about 400 IADR members, are expected.

The Dental Materials Group, composed of IADR members whose chief research interest is improvement of materials used in dentistry, will meet concurrently. About fifty research papers will be presented.

## Western Society of Periodontology

The Western Society of Periodontology announces that its eighth annual scientific meeting will be held at the Tropicana Hotel in Las Vegas, Nevada, March 19 to 21, 1961. The guest speaker will be Dr. Robert F. Moyers of the University of Michigan.

For further information, write Dr. Daniel Feder, 152 South Lasky Dr., Beverly Hills, California.

## University of Alabama

The School of Dentistry announces that a course in complete denture prosthesis will be given on March 10, 11, and 12, 1961, by Dr. William L. McCracken.

## Albert Einstein College of Medicine, Yeshiva University

The Albert Einstein College of Medicine, Department of Dentistry, will sponsor post-graduate courses in anesthesiology, endodontics, oral pathology, oral surgery, periodontia, and prosthetics during the spring of 1961. These courses are designed to be integrated

to the general dental practice. Lecture sessions will be held at the Albert Einstein College of Medicine, and the clinical demonstrations will be given at the Dental Clinic, Jacobi Hospital.

For further information and application, please write to Director, Postgraduate Dental Division, Albert Einstein College of Medicine, Eastchester Rd. and Morris Park Ave., Bronx 61, New York.

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## Georgetown University

The following courses will be offered in the winter and spring of 1961 by the School of Dentistry:

Microscopic Oral Pathology. Jan. 23 to 27, 1961. S. N. Bhaskar and G. W. Greene. Tuition, $150.00.

Malocclusion, Its Detection, Moderation, and Interception. Feb. 3 to 5, 1961. Faustin N. Weber and Herman M. Crowder. Tuition, $100.00.

Intravenous Office Anesthesia. March 7 to 11, 1961. Harold W. Krogh and Edward Driscoll. Tuition, $250.00.

Full Denture Construction. April 28 to 30, 1961. Victor Sears. Tuition, $75.00.

Further information may be obtained from the Registrar, Georgetown University School of Dentistry, 3900 Reservoir Rd., N.W., Washington 7, D. C.

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## Loyola University School of Dentistry (Chicago)

Applications are now being accepted by the Graduate Division of the School of Dentistry for graduate study in the field of oral surgery, leading to the Master of Science degree. Selection of students for 1961 will be made in March, 1961.

A nondegree, full-time, twelve-month course in oral surgery is also available for qualified dental graduates.

Further information and application forms may be obtained by writing to the Director of the Graduate Division, Loyola University School of Dentistry, 1757 W. Harrison St., Chicago 12, Illinois.

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## University of Pennsylvania

The School of Dentistry is offering the following postgraduate courses:

Applied Anatomy. Feb. 22 to 24, 1961. Harry Sicher. Prosthetic Relations of the Temporomandibular Joint. Jan. 16 to 20, 1961. Ernest R. Granger and associates.

Diagnosis and Treatment Planning. March 6 to 10, 1961. Lester W. Burket, Alvin L. Morris, and Roy T. Durocher.

Practical and Simplified Procedures in Crown and Bridge Prothesis. March 13 to 17, 1961. Charles B. Sceia.

Complete Denture Prosthesis. March 27 to 30, 1961. Vincent R. Trapozzano.

Endodontics. April 10 to 21, 1961. Louis I. Grossman and associates.

Further information may be obtained by writing to Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce St., Philadelphia 4, Pennsylvania.

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## Temple University School of Dentistry

An intensive participation course in surgical endodontic techniques will be presented March 6 to 10, 1961. The course is under the direction of Dr. Leonard Parris, assisted by

Drs. Harold Rappaport and Robert Uchin. Closed-circuit television will be utilized during the clinical demonstrations. Postgraduate students will carry out surgical endodontic procedures on clinic patients under the supervision of the teaching staff. Previous training in basic endodontics is a prerequisite for this course.

For further information, write Dr. Louis Herman, Director of Postgraduate Studies, Temple University School of Dentistry, 3223 North Broad St., Philadelphia 40, Pennsylvania.

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## Mount Sinai Hospital Oral Pathology Conference

The second clinical oral pathology conference of the season will be held at 4:30 P.M. on Friday, Jan. 20, 1961, in Lecture Room 3-B on the third floor of the Administration Building, Mount Sinai Hospital. The topic will be "Leukoplakia: A Re-evaluation of Concepts," and the speaker will be Dr. Lester R. Cahn.

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## University of Texas
## M. D. Anderson Hospital and Tumor Institute

"The Molecular Basis of Neoplasia" is the theme of the 1961 symposium on fundamental cancer research, sponsored by the University of Texas M.D. Anderson Hospital and Tumor Institute. Sessions for the fifteenth annual symposium were announced by Dr. Saul Kit, chairman of the meeting, for Feb. 23, 24, and 25, 1961.

The symposium, which annually evokes international interest, will consist of thirty-four presentations by scientists from the United Kingdom, Austria, Israel, and the United States.

A high light of the meeting will be the presentation of the Bertner Foundation Award, which is bestowed each year during the symposium for an outstanding contribution in the field of cancer research.

Programs and information on the meeting may be obtained by writing the Publications Department, University of Texas M.D. Anderson Hospital and Tumor Institute, Texas Medical Center, Houston 25, Texas.

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## Veterans Administration Medical Teaching Group Hospital

The Veterans Administration Medical Teaching Group Hospital of Memphis, Tennessee, announces that two rotating dental internships and one residency in prosthodontia will be available for the year beginning July, 1961.

Information and application forms may be obtained from the Chief, Dental Service, Veterans Administration Medical Teaching Group Hospital, Memphis, Tennessee.

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## Mount Sinai Hospital

The Department of Dental and Oral Surgery of the Mount Sinai Hospital announces that another in its series of Wednesday evening meetings will be held at 8:15 P.M. on Jan. 18, 1961, in Lecture Room 3-B on the third floor of the main building of the Mount Sinai Hospital, 100th St. and 5th Ave., New York, New York. The subject will be "Root Canal Therapy in the Concept of Modern Dentistry," and it will be presented by Dr. Morton Brenner and staff, of the Section on Endodontics.

A pre-meeting dinner will be served at 6:15 P.M. Members of the dental profession are welcome. Information and reservations may be obtained by calling TR 6-1000, extension 418 or 203.

## Position Open at Temple University School of Dentistry

Applications are now being accepted for the position of Research Fellow and Clinical Instructor in the Department of Endodontics at the Temple University School of Dentistry in Philadelphia.

The position offers the applicant an opportunity to pursue research projects related to endodontics, while at the same time instructing students in clinical endodontic techniques.

Remuneration is $5,200.00 a year. The applicant must be available Aug. 1, 1961.

Complete information as to training, interest, and clinical experience should be sent to Dr. Leonard Parris, Temple University School of Dentistry, Broad St. above Allegheny Ave., Philadelphia, 40, Pennsylvania.

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## University of Pennsylvania

Research and teaching traineeships in the basic sciences, in periodontology, and in the comprehensive treatment and rehabilitation of the cleft palate patient are available at the School of Dentistry, University of Pennsylvania.

Candidates for traineeships in the basic sciences must qualify for admission to the Graduate School of Arts and Sciences. These traineeships may lead to M.S. or Ph.D. degrees.

All traineeships are supported by the United States Public Health Service. Annual stipends vary from $4,500.00 and up, depending on the qualifications and needs of the successful candidates. Inquiries concerning the basic science and cleft palate traineeships should be sent to the Chairman, Committee on Traineeships and Fellowships, University of Pennsylvania, School of Dentistry, 4001 Spruce St., Philadelphia 4, Pennsylvania. Inquiries about the traineeships in periodontology should be sent to the Dean, Graduate School of Medicine, 237 Medical Laboratories, University of Pennsylvania, Philadelphia 4, Pennsylvania.

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## Tufts University School of Dental Medicine

The Division of Graduate and Post-Graduate Studies announces the following refresher courses:

Hospital Management of the Dental Patient. Jan. 11 to 13, 1961. Tuition, $100.00.

Construction of the Full Crown. Jan. 16 and 17, 1961. Tuition, $75.00.

Periapical Surgery. Jan. 26 and 27, 1961. Tuition, $75.00.

Orthodontic Principles of Interest to the General Practitioner. Each Wednesday for eight weeks, Feb. 8 to April 5, 1961. Tuition, $200.00.

Minor Oral Surgery. Each Wednesday for six sessions, March 1, 8, 22, and 29, April 5 and 12, 1961. Tuition, $175.00.

Crown and Bridge Construction. Feb. 15 and 16, 1961. Tuition, $75.00.

Hypnosis and Its Clinical Applications. March 16 to 22, 1961. Tuition, $200.00.

Endodontics for the General Practitioner. March 20 to 23, 1961. Tuition, $175.00.

Partial Denture Design and Construction. March 23 and 24, 1961. Tuition, $75.00.

Participation Course in Periodontology. April 24 to 28, 1961. Tuition, $175.00.

Clinical Adjustment of the Occlusion. May 3 to 5, 1961. Tuition, $125.00.

Surgical Management of Skeletal Deformities. May 8 to 10, 1961. Tuition, $125.00. (Limited to specialists and members of oral surgery societies.)

Diagnosis and Treatment of Pain and Other Symptoms Arising From Temporomandibular Joint Disturbances. May 23 and 24, 1961. Tuition, $75.00.

Full Denture Prosthesis (Participation Course). June 5 to 9, 1961. Tuition, $175.00.

For information about the above courses, please write to Director, Graduate and Post-Graduate Division, School of Dental Medicine, Tufts University, 136 Harrison Ave., Boston 11, Massachusetts.

# REVIEWS OF THE LITERATURE

## BOOK REVIEWS

**Dental Health Education.** Frances A. Stoll, R.D.H., Ed. D., Professor of Dental Hygiene and Director of Courses for Dental Hygienists, Columbia University, New York. Second edition. Philadelphia, 1960, Lea & Febiger. 253 pages, illustrated. Price, $5.50.

This monograph is written in an easy, straightforward style and is organized logically into a definition of the problem under consideration, dental health education.

The clear, well-integrated text is of value to dental health educators, dentists, dental hygienists, classroom teachers, and all others responsible for dental health instruction, service, and care programs. It is a highly practical guide to curriculum planning, a useful textbook for student dental hygienists.

*G. A. Coleman.*

**Oral Pathology.** Kurt H. Thoma, D.M.D., Dr. Med. Dent. H.C. (Zurich), F.D.S.R.C.S. (Eng.), Hon. F.D.S.R.C.S. (Edin.), F.A.C.D., and Henry M. Goldman, D.M.D., F.A.C.D. Fifth edition. St. Louis, 1960, The C. V. Mosby Company. 1523 pages, 1704 illustrations, including 111 in color. Price, $27.50.

This well-known text, which has served dentistry for many years, now boasts the addition of a distinguished collaborator. Dr. Henry Goldman's prominent position in the field of oral pathology complements that of the original author, Dr. Thoma. Dr. Goldman's contributions are particularly noticeable in the chapters dealing with oral medicine.

Although some changes have been made in the format, these in no way parallel in scope those occurring in the previous edition. Among the more notable changes has been the addition of numerous good color plates. The bibliographies, as in the past, are extensive and comprehensive. Illustrations are numerous and, on the whole, good. In particular, the photomicrographs exhibit a most satisfactory amount of detail.

This new edition is consistent with the traditions of the former ones and is recommended as highly as were the others. This is still the best text dealing with oral pathology.

*J. B.*

**Bone Homografts in the Surgical Treatment of Cysts of the Jaws and Periodontal Pockets.** Heyman Krømer, Institute for Experimental Medical Research, University of Oslo. Oslo, 1960, Oslo University Press. 185 pages, 64 photographs, tables. Price, Norw. Kr. 45.

This is an extremely interesting monograph dealing with the subject of homografts. The use of bone grafts in oral surgery depends on a thorough understanding of what happens during the healing process. The new bone is either formed by survival and proliferation of the transplanted cells or produced by the host cells induced by the transplant.

The author's investigation was carried out with the use of radioisotopes by which it is possible to determine whether or not the new bone originates from the surviving cells of the graft, from the covering and lining cells of the bone of the host bed, or from mesenchymal cells of the connective tissue.

The plan of the work was threefold: (a) operative treatment of cysts of the jaw with bone grafts; (b) operative treatment of periodontal pockets with bone grafts; and (c) experiments on dogs.

The monograph is well written and contains excellent illustrations. It is recommended for study by all who are interested in this field.

*K. H. T.*

**El Tercer Molar Inferior Retenido (The Retained Inferior Third Molar).** Guillermo A. Ries Centeno. Buenos Aires, 1960, Libreria "El Ateneo." 334 pages with 1380 illustrations.

The well-known author of *Chirugia Bucal* presents an excellent monograph on the clinical pathology, diagnosis, and surgery of the retained mandibular third molar. The book contains a prologue by Dr. Harry M. Seldin of New York and an introduction by the author. Dr. Ries Centeno states that an anatomic study is of greatest importance, and he devotes eighty-eight pages to the surgical and topographic anatomy investigated by means of dry specimens, sections, and x-ray pictures of the area in which the third molar is found. The blood supply and neurology, as well as the teeth themselves, are described, along with anatomic variations so common to these teeth.

The roentgen techniques giving the best information are taken up next, and a great number of x-ray pictures of the various positions are added. Unusual locations and various pathologic complications are included.

The surgery of the third molar is contained in the last 142 pages. The various techniques are beautifully illustrated by means of serial drawings as well as roentgenograms.

The author is to be congratulated. He has made available a large collection of cases with indication of methods of treatment. The publishers have produced a beautiful book, with illustrations of first-class quality.

*K. H. T.*

**Xylocaine: Chemistry, Pharmacology, and Clinical Applications.** Worcester, Mass., 1960, The Astra Pharmaceutical Products, Inc. 80 pages.

This is a useful monograph describing the chemistry of Xylocaine. It also reviews the clinical application of local anesthetics for surface anesthesia and injection for various fields of medicine and dentistry, including the newest and most interesting techniques of intravenous analgesia and anesthesia produced with local anesthetics.

Complications and sequelae related to the local anesthetic agents are described. Both systemic and local reactions receive consideration. An extensive bibliography is appended.

*K. H. T.*

**Strahlengefährdung und Strahlenschutz in der zahnärtlichen Röntgendiagnostik (Danger of and Protection Against Irradiation in Dental Roentgen Diagnosis).** Professor Theodor Kirsch, Dr. med., Dr. med. dent. Heidelberg, Germany, 1960, Alfred Hüthig Verlag. GMBH 59 pages, 47 illustrations. Price, DM. 6.80.

In the introduction the author points out that one can speak of a world-wide irradiation problem which concerns not only the profession but the entire population. Patients are entitled to know what the chances are when they submit to x-ray examinations recommended by the dentist. The dentist therefore should be informed, and this monograph has been written to furnish scientific information based on actual measurements. Also, the recommended protective measures are discussed and evaluated.

*K. H. T.*

**Synopsis of Pathology.** W. A. D. Anderson, M.A., M.D., F.A.C.P., F.C.A.P., Professor of Pathology, University of Miami School of Medicine, and Director of Pathology Laboratories, Jackson Memorial Hospital, Miami, Florida. Fifth edition. St. Louis, 1960, The C. V. Mosby Company. Price, $9.25.

Since the first edition of this book appeared in 1942, many interns have said, "We use Anderson's *Synopsis of Pathology* in our school." This is easily understood, for this handy, small volume is devoted to the basic aspects of disease processes.

The fifth edition has been rewritten in parts; for instance, new recognition is given many viruses with respect to the new fundamental knowledge of the nature of viruses.

The practical value of cytologic diagnosis of malignancy, carcinoma in situ, and precancerous changes are presented so that they may be better understood.

The revision of previously existing material and the selection of new material for this edition add to its importance as a textbook for the dental student.

*Thomas J. Cook.*

**Basic Periodontology.** A. Bryan Wade, B.Ch.D., F.D.S., R.C.S., Director, Department of Periodontology, Royal Dental Hospital of London School of Dental Surgery, University of London. Baltimore, 1960, Williams & Wilkins Company (exclusive U. S. agents). 303 pages, 223 illustrations. Price, $10.00.

This text is divided into four parts: fundamentals, health, disease, and treatment. The word "basic," according to the author, has been included in the title because this book does not profess to include all that is known about periodontal tissues.

The book is clearly written and well illustrated. There is no bibliography.

*R. A. Williams.*

**Endodontics.** Harry J. Healy, A.B., D.D.S., M.S.D., F.A.C.D., Chairman, Division of Endodontics, Indiana University School of Dentistry, Indianapolis, Indiana. St. Louis, 1960, The C. V. Mosby Company. 370 pages, 149 illustrations. Price, $7.75.

This publication of the Postgraduate Dental Lecture Series is in the handy pocket size which is proving very popular. It is artistically bound and printed, well illustrated, and easy to read.

The author was assisted by several members of his faculty in such fields as diagnosis, pathology, prescription writing, bleaching, surgery, and pedodontia. Many details of technique which have proved valuable to the author and his associates are included. The price places the book within the reach of the student and the practitioner.

*Thomas J. Cook.*